In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: May 21, 2025

SANDY M. FOUKARAKIS,	*	PUBLISHED
Petitioner,	*	No. 20-1547V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Dismissal; Influenza ("Flu") Vaccine; Transverse Myelitis ("TM"); Optic Neuritis.
Respondent.	*	

Phyllis Widman, Widman Law Firm, LLC, Linwood, NJ, for Petitioner.
Felicia Langel, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION[1]

I. INTRODUCTION

On November 9, 2020, Sandy M. Foukarakis ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018).[2] Petition (ECF No. 1). On December 16, 2020, Petitioner filed an amended petition alleging that the influenza ("flu") vaccine she received on November 8, 2018 caused Guillain-Barré syndrome ("GBS"); chronic inflammatory

[1] Because this Decision contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims' website and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018). All citations in this Decision to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

demyelinating polyradiculoneuropathy ("CIDP"), transverse myelitis ("TM"), as well as "[h]eadaches, loss of motor function, loss of bladder function, vision changes/optic neuritis, and/or other eye condition(s), and/or significantly aggravated[3] a condition." Amended ("Am.") Petition at Preamble (ECF No. 7). Respondent argued against compensation, stating that this petition "should be denied and the case should be dismissed." Respondent's Report ("Resp. Rept.") at 1, 14 (ECF No. 16).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards,[4] the undersigned finds that Petitioner has failed to provide preponderant evidence that her flu vaccine caused her TM, optic neuritis, or any other alleged conditions. Thus, Petitioner has failed to satisfy her burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, Petitioner is not entitled to compensation.

II. ISSUES TO BE DECIDED

The parties stipulated that Petitioner received a flu vaccine on November 8, 2018. Joint Sub. at 1. The parties do not dispute that the vaccine Petitioner received appears on the Vaccine Injury Table and the vaccination was administered in the United States. Id.

The parties dispute "the nature and diagnosis of Petitioner's alleged injuries." Joint Sub. at 1. In her prehearing brief, Petitioner argues that she suffers from a "demyelinating disease." Pet. Prehrg. Br. at 3. In her supplemental brief, Petitioner asserts that she suffers from central nervous system ("CNS") injury, specifically TM and optic neuritis. Pet. Suppl. Br. at 2, 5

[3] While Petitioner included "significantly aggravated" in her petition and her amended petition, Petitioner does not address significant aggravation in any of her expert reports. See Petitioner's Exhibits ("Pet. Exs.") 30, 43, 45, 110-11. Additionally, Petitioner's pre-hearing and supplemental briefs do not address significant aggravation or the Loving prongs. See Pet. Prehearing Brief ("Pet. Prehrg. Br."), filed Feb. 5, 2024 (ECF No. 93); Pet. Supplemental Br. ("Pet. Suppl. Br."), filed July 24, 2024 (ECF No. 120); see also Loving v. Sec'y of Health & Hum. Servs., 86 Fed. Cl. 135 (2009) (adopting a six-prong test for proving a significant aggravation claim). The parties' joint submissions only identify whether the flu vaccine "caused [Petitioner's] alleged injuries" as an issue to be resolved and do not address whether the flu vaccine significantly aggravated Petitioner's condition. Joint Prehearing Submission ("Joint Prehrg. Sub."), filed Feb. 26, 2024, at 1 (ECF No. 99); Joint Sub., filed July 24, 2024, at 1 (ECF No. 119). Accordingly, the undersigned does not address significant aggravation or the Loving prongs in this Decision.

[4] While the undersigned has reviewed all the information filed in this case, only those filings and records that are most relevant will be discussed. See Moriarty v. Sec'y of Health & Hum. Servs., 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision."); see also Paterek v. Sec'y of Health & Hum. Servs., 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

("Petitioner's symptoms and thus, diagnoses, were somewhat complex and overlapping . . . but they are nonetheless consistent with her CNS injury."). Respondent argues Petitioner has not established by preponderant evidence that she suffers from TM or optic neuritis, and further asserts that Petitioner has failed to show "that she was diagnosed with a compensable injury." Resp. Suppl. Br., filed Aug. 22, 2024 (ECF No. 121).

The parties also dispute the issue of "[w]hether the flu vaccine administered to [P]etitioner on November 8, 2018[] caused her alleged injuries." Joint Sub. at 1.

III. BACKGROUND

A. Medical Terminology

The following conditions are referenced in Petitioner's medical records.

1. Guillain-Barré Syndrome

GBS is "an acute monophasic peripheral neuropathy" that has four major subtypes characterized by differing clinical courses. 42 C.F.R. § 100.3(c)(15)(i). Common to these clinical subtypes, the "interval between the first appearance of symptoms and the nadir of weakness is between 12 hours and 28 days," followed by a "clinical plateau" and subsequent improvement "without significant relapse," although "[t]reatment related fluctuations" in symptoms may occur for up to nine weeks. Id.

The most common subtype of GBS is acute inflammatory demyelinating polyneuropathy ("AIDP") which is associated with "focal demyelination of motion and sensory peripheral nerves and nerve roots." 42 C.F.R. § 100.3(c)(15)(ii). There are five criteria for diagnosis of a Table GBS claim in the Vaccine Table. While this is not a "Table Claim" the criteria for diagnosis are informative as related to diagnosis. They include:

> (A) Bilateral flaccid limb weakness and decreased or absent deep tendon reflexes in weak limbs;
> (B) A monophasic illness pattern;
> (C) An interval between onset and nadir of weakness between 12 hours and 28 days;
> (D) Subsequent clinical plateau (the clinical plateau leads to either stabilization at the nadir of symptoms, or subsequent improvement without significant relapse; however, death may occur without a clinical plateau); and,
> (E) The absence of an identified more likely alternative diagnosis.

Id. at § 100.3(c)(15)(ii)(A)-(E).

2. Chronic Inflammatory Demyelinating Polyneuropathy

CIDP is a "slowly progressive, autoimmune type of demyelinating polyneuropathy" that occurs most commonly in young adults and is related to GBS. Chronic Inflammatory Demyelinating Polyneuropathy, Dorland's Med. Dictionary Online, https://www.dorlands

online.com/dorland/definition?id=99346 (last visited May 8, 2025). CIDP is characterized by "by progressive weakness and impaired sensory function in the limbs and enlargement of the peripheral nerves, usually with elevated protein in the cerebrospinal fluid [("CSF")]." Id. The presenting symptoms "often include tingling or numbness of the digits, weakness of the limbs, hyporeflexia or areflexia, fatigue, and abnormal sensations." Id.

3. Transverse Myelitis

TM is a rare condition "in which an immune-mediated process causes neural injury to the spinal cord, resulting in varying degrees of weakness, sensory alterations[,] and autonomic dysfunction." Pet. Ex. 81 at 1.[5] Autonomic symptoms include "increased urinary urgency, bowel or bladder incontinence, difficulty voiding, or bowel constipation." Resp. Ex. A-2 at 1;[6] see also Pet. Ex. 84 at 2.[7] TM affects "one or two segments and predominantly the white matter of the [spinal] cord." Pet. Ex. 31 at 6.[8] "When the inflammatory lesion extends across more than [three] vertebral segments longitudinally, it is commonly referred to as [longitudinally extensive TM]." Pet. Ex. 82 at 2;[9] see also Resp. Ex. B-3 at 1.[10]

The TM Consortium Working Group proposed uniform diagnostic criteria for acute TM:

1. Development of sensory, motor, or autonomic dysfunction attributable to the spinal cord
2. Bilateral signs and/or symptoms (though not necessarily symmetric)
3. Clearly defined sensory level
4. Exclusion of extra-axial compressive etiology by neuroimaging ([magnetic resonance imaging ("MRI")] or myelography; [computerized tomography ("CT")] of spine not adequate)

[5] N. Agmon-Levin et al., Transverse Myelitis and Vaccines: A Multi-Analysis, 18 Lupus 1198 (2009).

[6] Transverse Myelitis Consortium Working Group, Proposed Diagnostic Criteria and Nosology of Acute Transverse Myelitis, 59 Neurology 499 (2002).

[7] Douglas Kerr & Harold Ayetey, Immunopathogenesis of Acute Transverse Myelitis, 15 Current Op. Neurology 339 (2002).

[8] Dimitrios Karussis & Panayiota Petrou, The Spectrum of Post-Vaccination Inflammatory CNS Demyelinating Syndromes, 13 Autoimmunity Revs. 215 (2014).

[9] Wafa Akkad et al., Longitudinally Extensive Transverse Myelitis Following Vaccination With Nasal Attenuated Novel Influenza A(H1N1) Vaccine, 67 Archives Neurology 1018 (2010).

[10] Dean M. Wingerchuk et al., International Consensus Diagnostic Criteria for Neuromyelitis Optica Spectrum Disorders, 85 Neurology 177 (2015).

5. Inflammation within the spinal cord demonstrated by CSF pleocytosis[11] or elevated [Immunoglobulin ("Ig") G][12] index or gadolinium enhancement . . . and,
6. Progression to nadir between [four] h[ours] and 21 days following the onset of symptoms . . . .

Resp. Ex A-2 at 2 tbl.1. The TM Consortium Working Group noted that requiring objective inflammation of the spinal cord, such as CSF pleocytosis, may be a limitation of the proposed diagnostic criteria and may be "perceived as too restrictive." Id. at 3-4.

4. Optic Neuritis

Optic neuritis is "inflammation of the optic nerve" and a "common clinical manifestation of [CNS] inflammation." Resp. Ex. E-1 at 1.[13] Optic nerve inflammation may be caused by "autoimmunity, infection, granulomatous disease, paraneoplastic disorders, and demyelination caused by inflammatory or infectious disease." Id.

Optic neuritis typically presents as "acute, unilateral, painful vision loss." Resp. Ex. E-1 at 2. On examination, patients typically have "visual acuity lost, visual field loss, color deficient, and an afferent pupillary defect in the affected eye." Id.; see also Pet. Ex. 31 at 3. The extent of visual acuity loss can "vary significantly" and optic neuritis associated with neuromyelitis optica ("NMO") has "a lower probability of visual recovery." Resp. Ex. E-1 at 2, 25.

5. Neuromyelitis Optica

NMO is a "severe, demyelinating disease of the [CNS] that preferentially affects the optic nerve and spinal cord." Pet. Ex. 31 at 6; see also Resp. Ex. E-1 at 11; Resp. Ex. B-3 at 1. NMO is distinct from multiple sclerosis ("MS") and is associated with serum aquaporin-4 IgG

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[11] Pleocytosis is the "presence of a greater than normal number of cells in the [CSF]." Pleocytosis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=39556 (last visited May 8, 2025).

[12] Immunoglobulins are "structurally related glycoproteins that function as antibodies." Immunoglobulin, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=24894 (last visited May 19, 2025). IgA antibodies occur "at mucosal surfaces, in serum, and in secretions (saliva; tears; respiratory, genitourinary, and gastrointestinal tract secretions; colostrum), where it provides an early antibacterial and antiviral defense." Peter J. Delves, Molecular Components of the Immune System, Merck Manual, https://www.merckmanuals.com/professional/immunology-allergic-disorders/biology-of-the-immune-system/molecular-components-of-the-immune-system?query=acquired%20immunity (last visited May 19, 2025). IgM "is the first antibody formed after exposure to new antigen." Id. IgG antibodies are "the most prevalent" and the "primary [] Ig produced after re-exposure to antigen (secondary immune response)." Id.

[13] Jeffery L. Bennett, Optic Neuritis, 25 Continuum 1236 (2019).

antibodies ("AQP4 antibodies"). Pet. Ex. 31 at 6; Resp. Ex. B-3 at 1. Collectively, NMO disorders may be referred to as NMO Spectrum Disorders ("NMOSD"). Resp. Ex. B-3 at 1.

The International Panel for NMO Diagnosis ("IPND") proposed revised consensus diagnostic criteria for NMOSD with and without AQP4 antibodies. Resp. Ex. B-3 at 3. The diagnostic criteria for NMOSD without AQP4 antibodies require:

> 1. At least [two] core clinical characteristics[14] occurring as a result of one or more clinical attacks and meeting all of the following requirements:
>
> a. At least [one] core clinical characteristic must be optic neuritis, acute myelitis with [longitudinally extensive] TM, or area postrema syndrome;[15]
>
> b. Dissemination in space ([two] or more different core clinical characteristics);
>
> c. Fulfillment of additional MRI requirements, as applicable.
>
> 2. Negative tests for AQP4-IgG using best available detection method, or testing unavailable.
>
> 3. Exclusion of alternative diagnoses.

Id. at 3 tbl.1. The NMOSD diagnostic criteria are "are more stringent" for patients in whom AQP4 antibodies are not detected. Id. at 3. Patients with a positive test for AQP4 antibodies only need one core clinical characteristic and an exclusion of alternative diagnoses to meet the diagnostic criteria. Id. at 3 tbl.1.

B. Procedural History

Petitioner filed her petition on November 9, 2020. Petition. On December 16, 2020, Petitioner filed an amended petition, followed by medical records and Petitioner's affidavit in January 2021. Am. Petition; Pet. Exs. 1-25. Respondent filed a Rule 4(c) report on July 23, 2021, arguing against compensation. Resp. Rept. at 1. The case was then reassigned to the undersigned. Notice of Reassignment dated Aug. 20, 2021 (ECF No. 25).

Between December 2021 and April 2024, Petitioner continued to file medical records. Pet. Exs. 27-29, 40-42, 163-164. On March 29, 2022, Petitioner filed an expert report from Dr.

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[14] The core clinical characteristics of NMOSD include: (1) optic neuritis; (2) acute myelitis; (3) area postrema syndrome; (4) acute brainstem syndrome; (5) symptomatic narcolepsy or acute diencephalic clinical syndrome with NMOSD-typical diencephalic MRI lesions; (6) symptomatic cerebral syndrome with NMOSD-typical brain lesions. Resp. Ex. B-3 at 3 tbl.1.

[15] Area postrema syndrome is characterized by "intractable hiccups or nausea and vomiting." Michael C. Levin, Neuromyelitis Optica Spectrum Disorder (NMOSD), Merck Manual, https://www.merckmanuals.com/professional/neurologic-disorders/demyelinating-disorders/neuromyelitis-optica-spectrum-disorder-nmosd (last visited May 13, 2025). The area postrema is located "on the floor of the [fourth] ventricle" and "is a structure that controls vomiting." Id.

Georges Ghacibeh. Pet. Ex. 30. On July 29, 2022, Respondent filed expert reports from Dr. Mark Bromberg and Dr. Devin MacKay. Resp. Exs. A-B.

The undersigned held a Rule 5 conference on October 4, 2022. Order dated Oct. 6, 2022, at 1 (ECF No. 49). She was unable to provide preliminary findings due to the complexities of diagnosis. Id. The undersigned suggested the parties file additional expert reports addressing the issue of diagnosis as well as expert reports from an immunologist. Id. at 2. She requested an entitlement hearing if the parties were unable to settle the case. Id. at 3. On December 2, 2022, an entitlement hearing was set for March 26 through March 28, 2024. Prehearing Order dated Dec. 2, 2022 (ECF No. 52).

On February 2 and February 3, 2023, Petitioner filed an expert report from Dr. Omid Akbari and a supplemental expert report from Dr. Ghacibeh. Pet. Exs. 43, 45. On May 2, 2023, Respondent filed an expert report from Dr. William Hawse and supplemental expert reports from Dr. Bromberg and Dr. Mackay. Resp. Exs. C-E. On August 1, 2023, Petitioner filed additional supplemental reports from Dr. Ghacibeh and Dr. Akbari. Pet. Exs. 110-11. On October 24, 2023, Respondent filed the supplemental report of Dr. Hawse. Resp. Ex. E. Petitioner filed the final supplemental report of Dr. Akbari on December 13, 2023. Pet. Ex. 141.

In preparation for the March 2024 entitlement hearing, Petitioner submitted her prehearing brief on February 5, 2024 and Respondent submitted a prehearing brief on February 26, 2024. Pet. Prehrg. Br.; Resp. Prehrg. Br., filed Feb. 26, 2024 (ECF No. 96).

On March 18, 2024, the entitlement hearing was cancelled due to Petitioner's experts being unavailable on March 28, 2024, the third day of the hearing. Order dated Mar. 18, 2024 (ECF No. 104). The undersigned provided new hearing dates in July 2025. Id. at 2. The parties opted to submit the case for a ruling on the record in lieu of rescheduling the entitlement hearing. Joint Status Rept., filed Apr. 17, 2024 (ECF No. 106).

The parties requested to file supplemental briefs in support of the ruling on the record. Joint Status Rept., filed May 17, 2024 (ECF No. 110). On July 24, 2024, Petitioner filed a supplemental brief. Pet. Suppl. Br. Respondent filed a supplemental brief on August 22, 2024. Resp. Suppl. Br.

This matter is now ripe for adjudication.

C. Factual History

1. Summary of the Medical Records[16]

[16] This summary of medical records is taken from Respondent's Prehearing Brief, as the undersigned finds it provided an accurate representation of the relevant medical records. See Resp. Prehrg. Br. at 3-12. The undersigned has made some additions and edits.

Petitioner was forty-two years old and a physician assistant ("PA") at Northeast Spine and Sports Medicine when she received a flu vaccine in her right deltoid on November 8, 2018. Pet. Ex. 1; Pet. Ex. 11 at 9; Pet. Ex. 17 at 5.

Prior to her vaccination, Petitioner had a medical history significant for four recurrent episodes of infectious mononucleosis[17] and two miscarriages in the early-mid 2000s when she was found to be antinuclear antibody ("ANA") positive.[18] Pet. Ex. 8 at 452; Pet. Ex. 9 at 82; Pet. Ex. 10 at 120, 225; Pet. Ex. 16 at 3. Petitioner also had a familial history of a brother with rhabdomyolysis and a sister with vocal dyskinesia. Pet. Ex. 13 at 7; Pet. Ex. 15 at 20; Pet. Status Rept., filed Dec. 1, 2022, at 1 (ECF No. 50).

Petitioner's medical history following the flu vaccination on November 8, 2018 is as follows:

December 2018

Hospitalization at Ocean Medical Center (December 7-9, 2018).	December 7, 2018 – Petitioner complained of a December 3, 2018 onset of ascending paresthesias from feet to thighs without pain or weakness; saddle numbness; and slight paresthesias in the left arm. She reported a "recent flu vaccination 3 weeks ago." Examination showed intact motor function and reflexes.	Pet. Ex. 10 at 117-18, 122.
December 3, 2018 is twenty-five days post-vaccination.	Brain MRI (with contrast) showed scattered T2 enhancing lesions of unknown etiology; cervical MRI (with contrast) showed moderate canal stenosis; thoracic MRI (with contrast) unremarkable; lumbar MRI (no contrast) unremarkable.	Id. at 233-40.
	CSF normal; ANA negative; elevated ganglioside	Id. at 163-65, 173, 175-76, 183.

[17] Infectious mononucleosis is "a common, acute, usually self-limited, infectious disease caused by [Epstein-Barr virus ("EBV")] characterized by fever, membranous pharyngitis, lymph node and splenic enlargement, lymphocyte proliferation, and atypical lymphocytes; it gives rise to various immune reactions . . . [including] persistent antibodies to the virus." Infectious Mononucleosis, https://www.dorlandsonline.com/dorland/definition?id=89566 (last visited May 13, 2025).

[18] ANA are "antibodies directed against nuclear antigens." Antinuclear Antibodies, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56804 (last visited May 13, 2024). ANAs are "used to diagnose systemic lupus erythematous [("SLE")] and other autoimmune diseases." Antinuclear Antibody, Mosby's Manual of Diagnostic & Laboratory Tests 80 (6th ed. 2018).

	antibodies[19] (GM1=74, GM2=90; normal range is 1-50); elevated EBV[20] IgG >750 (normal range is 0-21.9); elevated EBV nuclear antigens elevated at 34.2 (normal range is 0-21.9); elevated myelin basic protein ("MBP") of 40.2 (normal range is 0-5.5).[21] Petitioner was treated with intravenous Solumedrol for three days.	
	Consulting neurologist Mary Sedarous, M.D., examined Petitioner. Assessment noted concern about saddle paresthesias and was "not supportive of a diagnosis of [GBS], especially in the context of intact motor responses and preserved reflexes in the lower extremities."	Id. at 117-18.
	December 8, 2018 – Petitioner complained of worsening symptoms in the left leg. Electromyography ("EMG") of the left leg was normal.	Pet. Ex. 10 at 223-25.
	December 9, 2018 – Examination showed left foot weakness.	Id. at 120-21.

[19] Gangliosides are "a group of a group of glycosphingolipids" that "occur predominantly in tissues of the [CNS]." Ganglioside, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=19729 (last visited May 13, 2025). Relatively high levels of ganglioside antibodies have been reported in GBS patients. Pet. Ex. 80 at 1 (A.A. Ilyas et al., Serum Antibodies to Gangliosides in Guillain-Barre Syndrome, 23 Annals Neurology 440 (1988)).

[20] EBV is "a virus of the genus Lymphocryptovirus that causes infectious mononucleosis." Human Herpesvirus 4, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=80849 (last visited May 8, 2025). EBV has also been identified as a "causative factor" of myelitis. Pet. Ex. 31 at 6.

[21] Myelin basic protein "constitutes about 30 per cent of myelin proteins; elevated levels of MBP occur in acute exacerbation of [MS] and acute cerebral infarction." Myelin Basic Protein, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=100535 (last visited May 12, 2025).

Hospitalization at Jersey Shore Medical Center (December 10-12, 2018).	December 10, 2018 – Petitioner complained of worsening leg weakness and difficulty walking. Examination showed 4/5 leg strength, reduced patellar reflexes, and reduced sensation from umbilicus down. EMG/nerve conduction study ("NCS") showed a "slight amplitude drop in motor studies and H waves" in the right more than the left leg, and was "[e]ssentially normal." December 11, 2018 – Petitioner's examination showed a generalized papular skin rash and slight weakness in the left leg. Consulting neurologist, Alan Deutsch, D.O., impression noted subjective ascending paresthesias "with normal objective EMG including F waves, normal CSF and MRIs. . . . There is no evidence of [TM], [MS], [GBS], or peripheral neuropathy on objective testing."	Pet. Ex. 9 at 94; Pet. Ex. 6 at 6. Pet. Ex. 9 at 83, 87. Id. at 86-87.
Office visits with neurologist Sandro Corti, M.D.	December 13, 2018 – Petitioner's examination showed numbness from T10 to her feet and leg areflexia. Dr. Corti wrote, "[Petitioner's] presentation is concerning for [GBS] due to active EBV." Referred to hospital for five days of intravenous immunoglobulin ("IVIG").[22] December 31, 2018 – Petitioner's rash and saddle numbness resolved with IVIG. Strength improved with physical therapy ("PT"). Petitioner complained of foot numbness and leg pain at night, and she reported working half days. Repeat EMG/NCS showed "decreased central recruitment in bilateral quadriceps and ankle dorsiflexors. The significance of this is unknown."	Pet. Ex. 3 at 30-31. Id. at 27; Pet. Ex. 6 at 6.

[22] IVIG is "a concentrated antibody-containing solution . . . [that] consists primarily of IgG" and is used to "treat or prevent severe bacterial and viral infections, autoimmune disorders, and immunodeficiency disorders." Margot L. Savoy, Passive Immunization, https://www.merck manuals.com/professional/infectious-diseases/immunization/passive-immunization (last visited May 13, 2025).

January 2019

Office visit with neuroimmunologist Marinos Dalakas, M.D.	January 8, 2019 – Petitioner reported walking unassisted since December 31, 2018, but with return of paresthesias (not as bad as before) from hip to feet on January 6, 2019. Examination showed normal cranial nerves and strength in arms, but reduced strength and sensation, and reduced patellar reflexes. Due to Petitioner's "relapsing paresthesias," Dr. Dalakas was concerned about CIDP and ordered monthly IVIG.	Pet. Ex. 11 at 9, 13-14.
Hospitalization at Monmouth Medical Center (January 12-16, 2019).	January 12, 2019 – Petitioner complained of recurrence of her ascending paresthesias, and she was admitted for four days of IVIG. January 14, 2019 – Repeat cervical MRI (with contrast) showed severe C5/6 stenosis with T2 signal suspicious for cord compression and edema; and repeat thoracic and lumbar MRIs (with contrast) were unremarkable. January 15, 2019 – The attending radiologist reviewed the cervical and thoracic MRIs and wrote an addendum: > There is a 7 mm T2 hyperintense lesion noted within the central cord at the T7-T8 level []. There is no evidence of cord expansion. Enhancement is questionable due to extensive motion artifact on the post contrast sequences. Diagnostic considerations would include a demyelinating plaque, such as those seen with [MS] or an inflammatory lesion such as focal myelitis. > C5-C6 level: There is a disc osteophyte complex that is causing mild compression of the central canal. There is no evidence of cord edema or expansion at this level.	Pet. Ex. 8 at 96-99. Id. at 206; Pet. Ex. 3 at 64. Pet. Ex. 8 at 209.
Follow-up with Dr. Corti.	January 31, 2019 – Petitioner reported that her symptoms were gradually improving after two rounds of IVIG, and she was still working. Assessment included acute TM, migraine, infectious mononucleosis and paresthesia and noted her TM was "most likely from EBV."	Pet. Ex. 3 at 21-22.

February 2019

	February 6, 2019 – Repeat EMG/NCS was normal. February 11, 2019 – Petitioner began taking medical cannabis for pain in her legs and feet.	Pet. Ex. 11 at 22, 53. Pet. Ex. 3 at 17.
Office visit with ophthalmologist Prinze Mack, M.D.	February 25, 2019 – Petitioner was referred to Dr. Mack to "rule out optic nerve disease, [TM]." Petitioner complained of a bilateral vision change that had "occurred gradually." Examination noted no afferent pupillary defects ("APD").[23] A scanning laser evaluation had "[f]indings consistent with optic nerve atrophy." However, "[v]isual field defect does in not correspond to nerve or retinal nerve fiber layer defect." Impression was "transient vision loss" in the right eye with the condition noted as "improved" and stable" as well as "optic [nerve] atrophy" in the right eye, myopia, and astigmatism.	Pet. Ex. 7 at 2-4.
Follow-up with Dr. Dalakas.	February 27, 2019 – Examination showed normal strength but reduced sensation in Petitioner's left leg and left side of trunk. Dr. Dalakas noted that the T7/8 lesion was "consistent with neuromyelitis" but not a neuropathy due to the normal EMG/NCS on February 6, 2019. Dr. Dalakas prescribed oral steroids, CellCept, and continued IVIG.	Pet. Ex. 11 at 35.
Follow-up with Dr. Corti.	February 28, 2029 – Petitioner reported "not much change" since her last visit. Assessment included acute TM "possibly from EBV."	Pet. Ex. 3 at 15-16.

[23] APD is a pupillary response tested using a swinging flashlight; an afferent defect is present if "the pupil paradoxically dilates when the flashlight swings to the side of the defect." Michael Rubin, Overview of Neuro-ophthalmologic and Cranial Nerve Disorders, Merck Manual, https://www.merckmanuals.com/professional/neurologic-disorders/neuro-ophthalmologic-and-cranial-nerve-disorders/overview-of-neuro-ophthalmologic-and-cranial-nerve-disorders#Diagnosis_v1042382 (last visited May 9, 2025). "Normally, the degree of pupillary constriction does not change as the flashlight is swung from eye to eye." Id.

March 2019

Follow-up with Dr. Mack.	March 12, 2019 – Examination showed "[p]ost inflammatory optic atrophy," and Dr. Mack "suspected optic nerve disease [in the right eye] due to [TM]." March 14-15, 2019 – Petitioner complained of worsening and changing symptoms with right arm numbness and an episode of urinary incontinence.	Pet. Ex. 7 at 5-6. Pet. Ex. 3 at 39; Pet. Ex. 6 at 3.

April 2019

Office visit with neurologist Carlos Pardo-Villamizar, M.D., at Johns Hopkins TM Center.	April 8, 2019 – Petitioner presented to Dr. Pardo-Villamizar for evaluation and a second opinion regarding her "neurological problems concerning for myelopathic syndrome." Petitioner complained of neck pain and "some pain in both eyes and poor vision in the left [eye]." Examination showed reduced reflexes in the arms and legs. Dr. Pardo-Villamizar noted that Petitioner's December 31, 2018 EMG "point[ed] against[] an axonal or demyelinating lesion of the bilateral lower extremities." His assessment was "thoracic myelopathy associated with mid thoracic cord lesion that appears to be centrally located based on the last two MRIs." Petitioner's current neurological examination appeared improved compared to December 2018 and January-February 2019 examination. He noted that "[t]he appearance of the lesion in the thoracic cord is unusual for a demyelinating disorder such as MS and even for NMO." He recommended a chest, abdominal, pelvic CT scan to check for "other systemic abnormalities such as enlarged lymph nodes or lung pathology that may suggest sarcoidosis, lymphomas[,] or granulomatous disorder." Dr. Pardo-Villamizar's primary diagnosis was thoracic myelopathy and noted "[s]ensory disease or syndrome and gait disturbance" were also "pertinent" diagnoses. April 17, 2019 – Repeat cervical MRI (with contrast) was unchanged; repeat thoracic MRI (with contrast) showed a non-enhancing lesion centrally at T7/8 that measured 4 x 3 x 11 mm, indicating an overall decrease in size as compared to January 14, 2019 (5 x 6 x 11 mm).	Pet. Ex. 6 at 1-7. Pet. Ex. 6 at 14-16.

Follow-up with Dr. Corti.	April 19, 2019 – Petitioner began tapering off oral steroids and stopped IVIG in preparation for a repeat CSF study.	Pet. Ex. 3 at 12.

May 2019 – *six months post-vaccination*

	May 3, 2019 – CT showed "[s]mall axillary and mediastinal lymph nodes."	Pet. Ex. 6 at 19.

June 2019

	June 12, 2019 – Positron emission tomography ("PET") scan showed "asymmetrical mild hypermetabolic activity within left axillary lymph nodes, likely reactive."	Pet. Ex. 6 at 23-24.
Follow-up with Dr. Corti.	June 14, 2019 – Petitioner reported good and bad days with 80% improvement. However, she was still having paresthesias, and she had developed muscle spasms. "At her last Hopkins visit, she had a PET scan. CT [] showed several enlarged lymph nodes. . . . [L]ymphoma is a possibility." Repeat lumbar punctures were "ordered to assess for neurosarcoid vs lymphoma."	Pet. Ex. 3 at 9-10.

July 2019

	July 3, 2019 – Repeat CSF study was normal. July 12, 2019 – Mammogram showed stable axillary lymph nodes when compared to 2014.	Pet. Ex. 3 at 46-53; Pet. Ex. 8 at 67. Pet. Ex. 20 at 11-12.

August 2019

Emergency Department visit to Ocean Medical Center.	August 16, 2019 – Petitioner complained of an itchy, diffuse rash following intravenous Solumedrol treatment. She was diagnosed with a drug reaction and discharged home.	Pet. Ex. 10 at 248-49.
Follow-up with Dr. Dalakas.	August 21, 2019 – Petitioner was recently treated for left arm numbness with three days of intravenous Solumedrol, but she had minimal improvement. She complained of distal and abdominal paresthesias and urinary urgency. Examination showed normal strength, reduced reflexes, and reduced sensation in forearms to hands, knees to feet, and at the T6/7 level. Diagnosis was myelitis with negative myelin	Pet. Ex. 11 at 51-55.

	oligodendrocyte glycoprotein ("MOG"), no NMO, and "[n]o history of optic neuritis." Given the recent exacerbation, repeat MRIs were ordered to "evaluate for new lesion of reactivation of prior lesion." Assessment was thoracic myelitis. Dr. Dalakas commented, "[w]hether her new sensory symptoms on the left arm are related to her reactivation of her thoracic lesion is unclear. . . . This is [an] autoimmune condition and she may relapse." The plan was to continue CellCept and consider rituximab if her myelitis was progressive. Petitioner was referred to a neuro-urologist. Dr. Dalakas also noted that Petitioner "planned to have an axillary lymph node biopsy to rule out CNS lymphoma."	

September 2019

Office visit with MS specialist and neurologist David Duncan, M.D.	September 4, 2019 – Petitioner presented to Dr. Duncan for further evaluation and a second opinion. Petitioner complained of right hand cramping. Examination showed leg weakness (left greater than right), reduced sensation in hands, feet, and at the T8/9 level, trace reflexes in the arms, no reflexes in the legs, and difficulty tandem walking. Dr. Duncan diagnosed petitioner with resolved GBS, an "incidental finding [of a] possible demyelinating lesion of unknown age" on thoracic MRI, and nonspecific demyelinating lesions in the brain. Although Dr. Duncan determined that Petitioner did not have MS, he wrote that she was "obviously at risk for [a] possible autoimmune demyelinating process." Dr. Duncan ordered more testing and recommended continuing IVIG and CellCept for "symptomatic management."	Pet. Ex. 15 at 18-21.
Office visit with urologist Mina Fam, M.D.	September 11, 2019 – Petitioner complained of nonpainful urinary urgency and frequency. Dr. Fam diagnosed Petitioner with neurogenic bladder with post-void residual.	Pet. Ex. 23 at 2, 7.
	September 14, 2019 – Autoimmune testing showed negative MOG antibody; negative AQP4 antibody; elevated glutamic acid decarboxylase 65 ("GAD-65") antibody of 24 (normal range is <5); elevated EBV IgG>750; EBV nuclear antigen>600 (normal range is 0-45); elevated Sjogren's antibody of 2.2 (normal	Pet. Ex. 15 at 23-33.

	range is <1.0);[24] John Cunningham Virus ("JCV") antibody positive.[25] September 23, 2019 – Urodynamic testing showed good muscle function with adequate bladder emptying.	Pet. Ex. 23 at 10, 18-23.
Office visit with gastroenterologist Vishal Jain, M.D.	September 25, 2019 – An incidental finding of a positive Hepatitis B surface antibody was "[l]ikely [a] previous exposure but no risk factors."	Pet. Ex. 14 at 5, 16.
Follow-up with Dr. Duncan.	September 30, 2019 – Petitioner reported "feeling notably better with increased strength," but she also had fatigue. Examination showed mild left leg weakness and trace reflexes, and Dr. Duncan diagnosed Petitioner with possible GBS, a "possible remote history of [TM]," and underlying CIDP. Petitioner did not "appear to qualify for a diagnosis of [MS], but does require continuing monitoring." Dr. Duncan prescribed rituximab as a replacement for CellCept and amantadine for fatigue.	Pet. Ex. 15 at 13-15.

October 2019

Follow-up with Dr. Corti.	October 16, 2019 – Examination showed that Petitioner had slightly reduced strength and sensation, and reduced reflexes in her legs. "Repeat MRI [was] mostly unchanged, [one] T2 lesion in left parietal region, raising concerns for MS." Dr. Corti noted that her left arm symptoms were due either to medication or cervical radiculopathy. Petitioner was again taking IVIG monthly and CellCept, but she reported that her symptoms returned three to four weeks after each IVIG treatment. Assessment	Pet. Ex. 3 at 3-4.

[24] Ro, La, and SS-C antibodies, i.e. Sjogren antibodies, are a subtypes of ANA that are "strongly associated with Sjogren syndrome" as well as other autoimmune diseases such as SLE, rheumatoid arthritis, and scleroderma." Anti-SS-A (Ro), Anti-SS-B (La), and Anti-SS-C Antibody, Mosby's at 88-89.

[25] JCV antibodies are used to "help identify individuals who have been exposed to the [John Cunningham] virus." Pet. Ex. 15 at 32. JCV is "often acquired during childhood. Most adults have been infected with the JC[V] but do not develop the disorder. The virus appears to remain inactive until something (such as a weakened immune system) allows it to be reactivated and start to multiply." Robyn S. Klein, Progressive Multifocal Leukoencephalopathy, Merck Manual, https://www.merckmanuals.com/home/brain-spinal-cord-and-nerve-disorders/brain-infections/progressive-multifocal-leukoencephalopathy-pml (last visited May 13, 2025).

	remained acute TM, migraine, infectious mononucleosis and paresthesia and noted "[TM] with whole body rash, possibly from EBV."	

November 2019 – *one year post-vaccination*

Office visit with rheumatologist Carrie Edelman, M.D.	November 5, 2019 – Dr. Edelman noted that Petitioner had "minimal symptoms since on IVIG" and four out of ten pain. Dr. Edelman also noted that Petitioner developed a neurogenic bladder after briefly discontinuing IVIG, and she ordered additional autoimmune labs.	Pet. Ex. 16 at 2-4.
Office visit with endocrinologist Krishna Chalasani, M.D.	November 25, 2019 – Petitioner reported fatigue, weight gain, and napping during the day, and Dr. Chalasani diagnosed her with Hashimoto's thyroiditis.[26]	Pet. Ex. 13 at 6-7, 10.

December 2019

	December 12, 2019 – Petitioner learned how to self-catheterize when having difficulty voiding.	Pet. Ex. 23 at 14, 24.

January 2020

Follow-up with Dr. Duncan.	January 15, 2020 – Dr. Duncan noted, "Overall physically [Petitioner] is improved and [has] been able to increase her activity level and was able to compete in a 5K race even though she had significant pain." Dr. Duncan also noted, "I do not feel that she carries a diagnosis of demyelinating disease of [the CNS] and did not recommend treatment related to her history of spinal cord abnormality but only follow-up." Dr. Duncan referred Petitioner to a neuromuscular specialist to determine whether to continue the IVIG and CellCept for her underlying CIDP. January 22, 2020 – Repeat brain MRI was unchanged compared to August 28, 2019.	Pet. Ex. 15 at 2-4. Id. at 11.

[26] Hashimoto's thyroiditis is "a progressive type of autoimmune thyroiditis with lymphocytic infiltration of the gland and circulating antithyroid antibodies; patients have goiter and gradually develop hypothyroidism." Hashimoto's Disease, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=70395 (last visited May 13, 2025).

February 2020

Follow-up with Dr. Edelman.	February 12, 2020 – Petitioner reported improved symptoms and that she had recently traveled to Costa Rica and was able to surf but not hike. Dr. Edelman noted that Petitioner's elevated GAD-65 antibody can be associated with stiff person syndrome.[27]	Pet. Ex. 16 at 7, 10.

July 2020

	July 6-9, 2020 – Repeat brain and cervical MRIs were unchanged compared to December 7, 2018. Repeat thoracic MRI showed "[n]o abnormal cord signal."	Pet. Ex. 9 at 391, 428, 463.
Emergency examination by Dr. Mack.	July 7, 2020 – Petitioner complained of right eye blurry vision, "objects moving horizontally," and eye "catching" with movement to the left. A vision evoked potential test was normal, and Dr. Mack noted, "odd there is no APD." Dr. Mack diagnosed Petitioner with right retrobulbar neuritis and oscillopsia,[28] and he prescribed oral steroids.	Pet. Ex. 7 at 8-10; Pet. Ex. 41 at 7.
Telehealth visit with Dr. Duncan.	July 22, 2020 – Dr. Duncan recommended that Petitioner continue her current treatment regimen of IVIG and CellCept, and he referred her to a neuromuscular specialist.	Pet. Ex. 29 at 96, 103.

September 2020

Telemedicine visit with GBS/CIDP specialist and neurologist Sami Khella, M.D.	September 8, 2020 – Petitioner complained that her hands contract into claws when touched, but that this improved with IVIG. Dr. Khella referred Petitioner to a neuro-ophthalmologist for evaluation of optic neuritis.	Pet. Ex. 19 at 6-7.

[27] Stiff person syndrome is "a condition of unknown etiology characterized by progressive fluctuating rigidity of axial and limb muscles in the absence of signs of cerebral and spinal cord disease but with continuous electromyographic activity; some cases have been linked to autoimmune conditions." Stiff Man Syndrome, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=111451 (last visited May 13, 2025). On October 19, 2022, Petitioner's work up for stiff person syndrome was negative. Pet. Ex. 41 at 8.

[28] Oscillopsia, also known as oscillating vision, is "a symptom in which objects appear to wiggle, jerk, or move back and forth." Oscillopsia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=35681 (last visited May 13, 2025).

October 2020

Office visit with neuro-ophthalmologist Madhura Tamhankar, M.D.	October 5, 2020 – Petitioner reported that, when she recently stopped taking IVIG due to an insurance issue, her right eye vision became blurry. Examination showed perfect vision in both eyes, normal Optical Coherence Tomography ("OCT"),[29] and trace APD in the right eye. Dr. Tamhankar wrote, "It is very unusual that she has had optic neuritis in the setting of IVIG taper," and she noted that Petitioner had no neuronal loss on OCT and no brainstem lesions on MRI. Dr. Tamhankar concluded that Petitioner had "a normal eye examination . . . and normal OCT parameters."	Pet. Ex. 25 at 3, 6, 10-11.
Follow-up with Dr. Khella.	October 8, 2020 – Petitioner's examination was normal, and Dr. Khella diagnosed her with a "possible demyelinating syndrome that responds to [IVIG]." However, he noted, "She has a paucity of radiographic findings, but does have a thoracic lesion - though I got [two] conflicting results from radiologists." October 20, 2020 – MRI orbits (with contrast) showed no optic neuritis and an unchanged (compared to March 18, 2019 brain MRI) probable right occipital cavernoma.[30] The radiologist also visualized the T7/8 signal abnormality and noted that it was unchanged compared to July 9, 2020.	Pet. Ex. 19 at 3-4. Pet. Ex. 28 at 8-9.
Follow-up with Dr. Duncan.	October 22, 2020 – The chief complaint noted Petitioner had a "history of [TM] [AIDP] question [CIDP] with history of nonspecific demyelination on MRI brain as well as negative CSF for oligoclonal bands." Petitioner's condition was "currently maintained on CellCept and pulse does of IVIG." Petitioner reported that she "feels good," but she had intermittent muscle spasms in her legs and right hand. Dr. Duncan noted Petitioner's "recurrent optical	Pet. Ex. 29 at 82-84, 90.

[29] OCT is "the creation of high-resolution (close to that of light microscopy) cross-sectional images of body structures by recording the reflection of infrared waves from the tissues." Optical Coherence Tomography, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=114047 (last visited May 9, 2025).

[30] Cavernoma is a "a vascular tumor composed mainly of large dilated blood vessels that often contain large amounts of blood." Cavernous Hemangioma, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=80327 (last visited May 13, 2025).

	symptoms of unclear etiology," but questioned optic neuritis. He noted "no evidence to make diagnosis of MS at this time." Recommended Petitioner continue current treatment regimen.	

November 2020 – *two years post-vaccination*

Telehealth visit with neurologist Noah Gilson, M.D.	November 12, 2020 –Petitioner discontinued CellCept in preparation for starting Rituxan.[31]	Pet. Ex. 27 at 12-14.

December 2020

Follow-up with Dr. Gilson.	December 29, 2020 – Petitioner reported being 85-90% recovered from her TM, having TM as a child, working part-time, and using medical cannabis for intermittent pain. Dr. Gilson noted, "The initial event followed a flu shot by 3 weeks." Dr. Gilson also noted that Petitioner "present[ed] a diagnostic dilemma as far as the definitive diagnosis" was concerned. However, Dr. Gilson recommended that she "hold off on getting any sort of immunization because she has had [TM]."	Pet. Ex. 27 at 9-10, 20.

March 2021

Follow-up with Dr. Gilson.	March 12, 2021 – Petitioner developed a skin rash following receipt of intravenous Solumedrol, and her first Rituxan infusion was stopped.	Pet. Ex. 27 at 6-7.
Follow-up with Dr. Duncan.	March 23, 2021 – Petitioner complained of "occasional right and left hand spasms," and she had been seen by neuromuscular specialist Dr. Sedarous. Dr. Sedarous diagnosed Petitioner with benign fasciculation syndrome. Dr. Duncan wrote, "We discussed [that Petitioner] has no clear diagnosis at this time and no specific therapy is recommended however continued close clinical and radiographic observation should continue." Dr. Duncan recommended discontinuing the IVIG, not starting Rituxan, and "focus[ing] on primarily symptomatic management."	Pet. Ex. 29 at 72-73, 77.

[31] Rituximab, the generic form of Rituxan, is a "a chimeric murine/human monoclonal antibody that binds the CD 20 antigen" and is "administered intravenously." Rituximab, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=43977 (last visited May 13, 2025).

September 2021

Follow-up with Dr. Gilson.	September 2, 2021 – Petitioner complained of episodic hand spasms and pain that did not respond to the medical cannabis. She was still working. Dr. Gilson diagnosed Petitioner with acute TM, maybe not NMO, and possibly stiff person syndrome[32] for which he prescribed Valium.	Pet. Ex. 27 at 3-4.
Follow-up with Dr. Duncan.	September 21, 2021 – Petitioner reported being hospitalized in May 2021 for acute onset weakness. She complained of generalized weakness and diffuse muscle and joint pain. Dr. Duncan again recommended discontinuing IVIG therapy.	Pet. Ex. 29 at 7, 19.

December 2021 – *three years post-vaccination*

	December 7, 2021 – Repeat testing for GAD-65 anti-body was negative. December 30, 2021 – Petitioner complained to her primary care provider of severe arm weakness. She was to have a repeat EMG/NCS and a nerve biopsy.	Pet. Ex. 40 at 6-7 Id. at 4.

February 2022

	February 7, 2022 – Repeat brain and cervical MRIs (no contrast) showed no change compared to July 6-9, 2020; MRI of T-spine showed "[s]mall focal cord signal abnormalities at the T7-T8 level and at the T10 level could represent areas of demyelination compared to [July 9, 2020]."	Pet. Ex. 41 at 11;
Follow-up with Dr. Gilson.	February 22, 2022 – Petitioner presented with a complaint of several days of severe jaw pain. History of present illness noted Petitioner "carries a diagnosis of possible MS versus NMO spectrum disease." Dr. Gilson noted that Dr. Duncan and Petitioner's "doctor at Jefferson" believe her condition "is actually MS." Past medical history included "GBS and possibly [TM] probably MS[,]" "EBV positive [t]iters[,]" and "[p]ossible [TM] as a child." Dr. Gilson's	Pet. Ex. 42 at 2-4.

[32] Petitioner later had a negative work up for stiff person syndrome. Pet. Ex. 41 at 8; see supra note 27.

	assessment was trigeminal neuralgia,[33] MS, acute TM, NMO ("may or may not have this"), and "other muscle spasm" possibly "some variant of stiff person syndrome." He prescribed oral steroids and referred Petitioner to neuromuscular specialist.	
Follow-up with Dr. Duncan.	February 28, 2022 – Petitioner complained of continued muscle spasms in hands and feet, and jaw pain from trigeminal neuralgia. Stopped chronic immunotherapy in October 2021.	Pet. Ex. 41 at 11.

March 2022

Follow-up with Dr. Duncan.	March 22, 2022 – History of present illness noted "recurrent EBV infections." Recommended Petitioner considers a second opinion from Dr. Newsom at Johns Hopkins.	Pet. Ex. 41 at 41-46.

June 2022

	June 11, 2022 – Repeat thoracic MRI (with contrast) showed "[s]table demyelinating plaques" at T7/8 with no new enhancement.	Pet. Ex. 41 at 18.

October 2022

	October 11, 2022 – Repeat lab testing MOG negative.	Pet. Ex. 41 at 18.
Follow-up with Dr. Duncan.	October 19, 2022 – Petitioner complained of acute eye and neurologic symptoms on June 1, 2022, and had a follow-up visit with an ophthalmologist. Examination showed no weakness and trace reflexes throughout. History of present illness noted a "presumed history of [GBS] and [TM]" as well as "?optic neuritis . . . cervical spinal stenosis, neuropathy . . . [and] recurrent [EBV] infections." Dr. Duncan noted that Petitioner discontinued IVIG in October 2021, and he recommended subcutaneous IVIG for "continued clinical worsening." Dr. Duncan wrote, "There is no evidence to make the diagnosis of [MS] at this time," and "At present time no clear diagnosis."	Pet. Ex. 41 at 7-10, 12, 18.

[33] Trigeminal neuralgia is "severe, episodic pain in the area supplied by the trigeminal nerve." Trigeminal Neuralgia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=92499 (last visited May 13, 2025).

November 2022 – *four years post-vaccination*

	November 21, 2022 – Repeat brain MRI (no contrast) "mild degree of patchy elevated signal in cerebral white matter concordant with the history of MS." No evidence of active demyelination. Stable.	Pet. Ex. 40 at 1-2.

2. Petitioner's Affidavits

Petitioner filed two affidavits. Pet. Exs. 2, 26. In her first affidavit, executed January 16, 2021, Petitioner averred she received the flu vaccine on November 8, 2018 and in December 2018, she "woke up and was unable to feel [her] legs. Pet. Ex. 2 at ¶¶ 3-4. She was subsequently hospitalized on two occasions, and given diagnoses of "weakness," GBS, CIDP, and finally TM "with complete 7 mm lesion on [her] thoracic spine." Id. at ¶¶ 4-10. Petitioner explained that prior to receiving the November 8, 2018 flu vaccine, she was "a healthy active individual—a triathlete." Id. at ¶ 11. As of January 16, 2021, Petitioner avers she has permanent cord damage, a limp, unsteadiness and numbness, and needs assistance walking. Id.

In her second affidavit, executed July 20, 2021, Petitioner addressed physical therapy records. Pet. Ex. 26 at ¶¶ 2-7. Petitioner explained that she received physical therapy at Northeast Spine medicine where she was employed. Id. at ¶¶ 2-3. For various reasons, her physical therapy encounters occurred offsite, were "non-billable," or occurred during "spare time," so no encounter notes or progress notes were documented. Id. at ¶¶ 3-7.

D. Expert Reports

1. Petitioner's Expert, Dr. Georges A. Ghacibeh, M.D.[34]

a. Background and Qualifications

Dr. Ghacibeh is the chief of neurology at Pascack Valley Medical Center and is an assistant professor in the department of neurology at Seton Hall-Hackensack Meridian School of Medicine. Pet. Ex. 38 at 1. He is board certified in neurology, clinical neurophysiology, sleep medicine, and epilepsy by the American Board of Psychiatry and Neurology. Id. at 2. He has additional certifications in Video-EEG Monitoring from the American Board of Clinical Neurophysiology and CT/MRI from the American Society of Neuroimaging. Id. He received his M.D. from Lebanese University in Beirut. Id. at 1. He subsequently completed a neurology residency at New York University and Bellevue Medical Center. Id. He then completed fellowships in behavioral and cognitive neurology and in clinical neurophysiology, epilepsy, and sleep medicine at the University of Florida. Id. Dr. Ghacibeh has lectured on and authored, or co-authored, publications on topics related to neurology, epilepsy, and clinical neurophysiology. Id. at 3-6.

[34] Petitioner submitted three expert reports from Dr. Ghacibeh. Pet. Exs. 30, 43, 111.

b. Opinion

i. Diagnosis

Dr. Ghacibeh opined Petitioner had TM with a "plausible diagnosis of [NMO]." Pet. Ex. 111 at 4. He acknowledged that Petitioner's clinical presentation was complex and noted that her diagnosis "remained somewhat elusive and controversial." Pet. Ex. 30 at 4.

In his first report, Dr. Ghacibeh opined that retrospectively "the most plausible diagnosis" was NMO based on Petitioner's T7 thoracic spine lesion, her fully reversible episode of optic neuritis, and her initial clinical presentation. Pet. Ex. 30 at 4. He opined Petitioner's initial clinical presentation of ascending sensory symptoms starting in her feet and extending to "a sensory level in the abdomen," along with Petitioner's ataxic[35] gait was consistent with NMO. Id. He noted that NMO is typically associated with AQ4P-IgG and MOG antibodies. Id. These antibodies were not present in Petitioner and Dr. Ghacibeh acknowledged this made her NMO diagnosis "atypical." Id. In his second and third reports, Dr. Ghacibeh maintained NMO as a "plausible diagnosis." Pet. Ex. 43 at 5; Pet. Ex. 111 at 3.

Dr. Ghacibeh opined that a diagnosis of TM was supported by a "well documented lesion in the thoracic spine associated with symptoms and examination findings that correlate with this lesion, including sensory symptoms in the lower extremities, a sensory level, bladder dysfunction, and [] possible mild weakness and gait dysfunction, in addition to a reversible episode of optic neuritis." Pet. Ex. 43 at 5; Pet. Ex. 111 at 3. He further emphasized "the certainty of the diagnosis of TM." Pet. Ex. 111 at 3.

Addressing Respondent's expert Dr. Bromberg's diagnostic concerns, Dr. Ghacibeh opined that inconsistency in Petitioner's physical examinations over time could be explained by "subtle signs" being missed by "less thorough examiners." Pet. Ex. 43 at 5. In his third report, Dr. Ghacibeh clarified that he was not discounting the accuracy of neurological examinations performed by Petitioner's treating neurologists, rather he explained that the basic neurological examination was a standard screening tool and more subtle abnormalities may not be tested for or noticed based on the results of the basic neurological examination. Pet. Ex. 111 at 2.

Regarding the lack of motor symptoms, Dr. Ghacibeh agreed with Dr. Bromberg that Petitioner's lesion involved the central cord which means it did not involve motor pathways. Pet. Ex. 43 at 2. He explained that the absence of motor symptoms does not exclude a diagnosis of TM as lesions in the spinal cord may involve only sensory fibers resulting in isolated sensory symptoms without motor symptoms. Id. Further, Petitioner's "isolated sensory systems and sensory findings on examination" were a common clinical presentation of a spinal demyelinating

[35] Ataxic gait is "an unsteady, uncoordinated walk, with a wide base and the feet thrown out, due to some form of ataxia." Ataxic Gait, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=77913 (last visited May 8, 2025). Ataxia is the "failure of muscular coordination" or "irregularity of muscular action." Ataxia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=4630 (last visited May 8, 2025).

disorder. Pet. Ex. 111 at 2. And her symptoms of gait disturbance, mild leg weakness, and bladder dysfunction are "easily explained by a spinal cord lesion." Id.

He opined that Petitioner's MRI showing a T7 thoracic spine lesion explained—both physiologically and anatomically—Petitioner's bladder dysfunction. Pet. Ex. 111 at 3. He noted Petitioner's bladder dysfunction is evidenced by her urinary symptoms and urodynamic studies showing a post void residual as well as her need to self-catheterize. Id.; Pet. Ex. 43 at 3. Petitioner's continued use of self-catheterization demonstrated persistent bladder dysfunction. Pet. Ex. 43 at 3.

Responding to Dr. Bromberg's other diagnostic concerns, Dr. Ghacibeh asserted that restriction of Petitioner's spinal lesion to a single level does not refute a diagnosis of TM. Pet. Ex. 43 at 2. A review of 1001 patients with confirmed TM by Abbatemarco et al.[36] found only 24.3% of patients had involvement of three or more spinal segments, i.e., longitudinally extensive TM. Id. (citing Pet. Ex. 44 at 4). While Abbatemarco et al. do not identify the percentage of patients with lesions limited to a single level, Dr. Ghacibeh asserted that around 70% of patients had lesions involving less than three segments, and presumably some of these had lesions limited to a single segment. Pet. Ex. 111 at 2. Dr. Ghacibeh concluded that "small lesions that are limited to small number of segments often lead to unequivocal symptoms of TM." Id.

Additionally, Dr. Ghacibeh opined that "the absence of elevated CSF proteins does not exclude [a TM] diagnosis." Pet. Ex. 43 at 3. In support, he cited Abbatemarco et al. who reported CSF protein levels ranged from 16 to 709 (normal reference range < 50 mg/dL) and were elevated in 68.4%, but not all, patients with TM. Id. at 2-3 (citing Pet. Ex. 44 at 4, 4 tbl.2).

Finally, Dr. Ghacibeh explained that Petitioner's "diagnostic uncertainty" at the onset of her symptoms did not rule out TM and noted that Petitioner presented with acute neurological symptoms. Pet. Ex. 111 at 3. He disagreed with Dr. Bromberg's argument that Petitioner did not have progression to nadir within four hours to 21 days after onset in light of Petitioner's "early misdiagnosis and the various treatment interventions that [Petitioner] had received which most likely impacted the course of the illness." Pet. Ex. 43 at 3.

Next, Dr. Ghacibeh opined that a diagnosis of optic neuritis is "very plausible." Pet. Ex. 111 at 3. He based his opinion on Dr. Mack's diagnosis of optic neuritis, Petitioner's clinical presentation and "initial objective examination," and the "overall clinical picture." Pet. Ex. 43 at 4; Pet. Ex. 111 at 3.

Dr. Ghacibeh noted Petitioner developed painful vision loss with an examination by Dr. Mack revealing "right optic atrophy secondary to right optic nerve damage," leading to a diagnosis of retrobulbar optic neuritis. Pet. Ex. 30 at 3. Examination by a second ophthalmologist was normal and MRI of the orbits was reported normal. Id. While Dr.

[36] Justin R. Abbatemarco et al., Modern Look at Transverse Myelitis and Inflammatory Myelopathy, 8 Neurology Neuroimmunology & Neuroinflammation e1071 (2021). This article was also filed as Resp. Ex. A-3.

Ghacibeh acknowledged that Petitioner's later testing was "normal," he opined the later normal testing and examination did not exclude or refute Petitioner's earlier diagnosis of optic neuritis. Pet. Ex. 43 at 4; Pet. Ex. 111 at 3. Rather, it demonstrated that Petitioner had an episode of optic neuritis that was "relatively mild and fully reversable." Pet. Ex. 43 at 4.

Further, he felt that Petitioner's entire clinical picture, "including presenting symptoms, Dr. Mack's examination," and the presence of a spinal demyelinating lesion, made an alternative diagnosis for Petitioner's optical symptoms "very unlikely." Pet. Ex. 111 at 3.

Regarding other diagnoses, Dr. Ghacibeh declined to "completely rule out" a diagnosis of GBS, but he thought it was "less certain" since Petitioner did not have typical CSF and electrophysical findings associated with GBS. Pet. Ex. 30 at 4. In his first report, he opined that it was "possible" Petitioner had "atypical GBS" and subsequently developed TM. Id. He also concluded that Petitioner's clinical presentation "argue[d] against a diagnosis of MS." Id. Abbatemarco et al., who conducted a large-scale retrospective review of 1,001 TM cases, reported that "MS was the final diagnosis" in 164 (16.7%) of these of cases. Pet. Ex. 44 at 4. Additionally, 50.86% of patients who had an abnormal brain MRI were eventually diagnosed with MS. Id.

ii. Althen Prong One

Dr. Ghacibeh explained that TM and other neurological conditions can be caused by vaccination. Pet. Ex. 30 at 5. He noted that there are "several medical theories causally connecting vaccination with immune dysfunction" and that these theories center around cross reactivity between antigens in the vaccine and antigens in the body. Id. He invoked molecular mimicry as a causal mechanism and explained that it occurs "when the immune system mistakes an antigen that normally belongs to the body as foreign and therefore, mounts an immune attack against it," resulting in autoimmunity. Id.

A second mechanism offered by Dr. Ghacibeh included "[o]verstimulation" and "alteration" of the immune system caused by "chemicals in the vaccine." Pet. Ex. 30 at 5. He did not identify any chemicals or substances in the vaccine which could have caused this purported immune system reaction.

Dr. Ghacibeh opined that medical literature provided a well-established relationship between various vaccines and the development of demyelinating diseases such as TM, optic neuritis, NMO, acute disseminated encephalomyelitis ("ADEM"),[37] encephalitis, and MS. Pet. Ex. 30 at 4-5 (citing Pet. Exs. 31-37).

[37] ADEM is "an acute or subacute encephalomyelitis or myelitis characterized by perivascular lymphocyte and mononuclear cell infiltration and demyelination; it occurs most often after an acute viral infection, especially measles, but may occur without a recognizable antecedent." Acute Disseminated Encephalomyelitis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=73033 (last visited May 8, 2025). ADEM is "believed to be a manifestation of an autoimmune attack on the myelin of the [CNS]." Id.

In Karussis and Petrou, the authors analyzed 71 cases of CNS demyelinating syndromes—including TM, optic neuritis, and NMO—that followed flu, human papilloma virus ("HPV"), hepatitis A and B ("Hep A" and "Hep B"), rabies, measles, rubella, yellow fever, anthrax, meningococcus, and tetanus vaccinations. Pet. Ex. 31 at 1. The authors concluded that "while there's no absolute way to definitely link the onset or exacerbation of demyelination with the vaccine[s]" the "close temporal association with the time of vaccination strongly argues in favor of such pathogenetic correlation." Id. at 7. The authors identified "[i]mmune adjuvants that are included in the vaccine preparations" and molecular mimicry as the main immunopathogenic mechanisms of post-vaccination CNS demyelination. Id. Dr. Ghacibeh did not provide evidence that the flu vaccine here contained an adjuvant. Moreover, the authors noted that "the most common type of myelitis worldwide is infectious myelitis" due to viruses or bacteria. Id. Causative viruses included EBV. Id.

Additional articles cited by Dr. Ghacibeh included Poser,[38] where the author asserted "neurological complications of vaccinations constitute an anatomical and pathological spectrum which may involve any and all parts of the central, peripheral[,] and autonomic nervous systems." Pet. Ex. 32 at 1. The author identified 41 case reports of neurological complications other than GBS following the 1956-1980 flu vaccines. Id. at 4 tbl.1. The onset of the neurological complications ranged from 30 minutes to 35 days after vaccination. Id. The author also identified 26 instances of neurological complications other than GBS following the 1976 swine flu vaccine. Id. at 2.

In Huynh et al.,[39] the authors performed a literature review of post-vaccination ADEM. Pet. Ex. 33 at 2. The authors described a case report of a patient presenting with bilateral optic neuropathy within three weeks of a flu vaccine followed by delayed onset of ADEM three months post-vaccination. Id. at 8-9. Of note, the authors reported that "post-infectious ADEM is associated with a preceding or concomitant infection that is most commonly viral." Id. at 4. EBV was identified as one of the causal viruses. Id.

Austin et el.[40] reported a case of TM following flu A (H1N1) vaccine. Pet. Ex. 34 at 1. The authors posited that vaccine adjuvants (primarily aluminum salts) cause autoimmunity through "molecular mimicry, epitope spreading, up-regulation of cytokines, and polyclonal activation of B and T lymphocytes." Id. However, Dr. Ghacibeh did not provide evidence that there was an adjuvant in the flu vaccine at issue here.

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[38] Charles M. Poser, Neurological Complications of Swine Influenza Vaccinations, 66 Acta Neurologica Scandinavica 413 (1982).

[39] William Huynh et al., Post-vaccination Encephalomyelitis: Literature Review and Illustrative Case, 15 J. Clinical Neurosci. 1315 (2008).

[40] Adam Austin et al., Transverse Myelitis Activation Post-H1N1 Immunization: A Case of Adjuvant Induction?, 17 IMAJ 120 (2015).

The study by Langer-Gould et al.[41] focused on whether Hep B and HPV vaccines increased the risk of MS or other CNS demyelinating syndromes. Pet. Ex. 35 at 1. The authors found "[v]accination of any type was associated with an increased risk of CNS [acute demyelinating syndrome] onset within the first 30 days after vaccination only in younger (< 50 years) individuals."[42] Id. But there was "no longer-term association of vaccines" with MS or CNS demyelinating syndromes. Id. They concluded that a "short-term increase in risk suggests that vaccines may accelerate the transition from subclinical to overt autoimmunity in patients with existing disease." Id. A history of infectious illnesses was present in 90 (11.5%) cases, and the case patients were "more likely to have had a visit for an infectious illness in the [six] months before symptom onset" compared to the control group. Id. at 3, 4 tbl.1.

Dr. Ghacibeh also noted case reports of TM following COVID-19 vaccination. Pet. Ex. 30 at 5. He cited Erdem et al.,[43] who also reported that "[TM] usually occurs as a post-infectious complication and appears to result from an autoimmune process." Pet. Ex. 39 at 1-2.

iii. Althen Prong Two

Dr. Ghacibeh opined that Petitioner's TM and subsequent optic neuritis were "complications of the [flu] vaccine she received on November 8, 2018." Pet. Ex. 30 at 5; Pet. 43 at 5; Pet. Ex. 111 at 3.

Dr. Ghacibeh opined that there was a "clear logical sequence of events between exposure to the vaccine and the development of immune-related illness." Pet. Ex. 30 at 5. Dr. Ghacibeh asserted that the "absence of typical antibodies associated with NMO" provided evidence that Petitioner's NMO "likely related to an exogenous immune trigger, such as a vaccine." Id. Moreover, Dr. Ghacibeh opined the "timeline is strongly supportive of the causative role of the vaccine." Pet. Ex. 111 at 4.

Responding to Dr. Mackay's criticism, Dr. Ghacibeh acknowledged that the cases of post-vaccination CNS inflammatory disorders referenced in his medical literature described clinical presentations different than the clinical presentation Petitioner experienced. Pet. Ex. 43 at 4. However, Dr. Ghacibeh argued that these differences in clinical presentation "provide[d] further support" that Petitioner's condition was "likely caused by vaccination" as the cited cases "prove[] that post-vaccination CNS inflammatory disorders are not a homogenous set of conditions, and can, therefore, cause different clinical presentations in different patients." Id.

Regarding EBV as an etiology for Petitioner's demyelinating illness, Dr. Ghacibeh noted that "the majority of the population has antibodies against EBV," thus he asserted the presence of

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[41] Annette Langer-Gould et al., Vaccines and the Risk of Multiple Sclerosis and Other Central Nervous System Demyelinating Diseases, 71 JAMA Neurology 1506 (2014).

[42] Petitioner was 42 years old at the time of her vaccination. Pet. Ex. 2 at ¶¶ 2-3.

[43] Nazan Simsek Erdem et al., Acute Transverse Myelitis After Inactivated Covid-19 Vaccine, 74 Ideggyogyaszati szemle 273 (2021).

EBV antibodies should be "considered inconsequential." Pet. Ex. 43 at 3. Citing to Abbatemarco et al., Dr. Ghacibeh noted the authors found that 3.3.% of TM cases were preceded by vaccination. Id. at 5 (citing Pet. Ex. 44 at 4). The Abbatemarco at el. study also found that 9.7% of TM cases were preceded by infection. Pet. Ex. 44 at 4. Dr. Ghacibeh opined that number of TM cases preceded by vaccination "seem[] to be a much higher percentage than the few case reports of EBV associated with TM." Pet. Ex. 43 at 5 (citing Pet. Ex. 44 at 4). Dr. Ghacibeh concluded, given Petitioner's clinical presentation, vaccination was a "much more plausible explanation." Id.

iv. Althen Prong Three

Dr. Ghacibeh's chronology of events showed Petitioner received the flu vaccination on November 8, 2018 and onset of sensory symptoms in her feet began December 3, 2018. Pet. Ex. 30 at 3-4. He opined that "the temporal relationship between receiving the [flu] vaccine and the development of sensory symptoms in her feet support a causal relationship between the vaccine and [Petitioner's] illness." Id. at 5. He explained that the "timeline" of Petitioner's case is consistent with reports that "acute central demyelination typically occurs within 30 days of vaccinations." Id. (citing Pet. Ex. 31). In Karussis and Petrou, the authors analyzed 71 cases of CNS demyelinating syndromes—including TM, optic neuritis, and NMO—that followed vaccinations. Pet. Ex. 31 at 1. The mean onset of symptoms following vaccination was 14.2 days. Id. Following the flu vaccine, the earliest onset of symptoms was five days, and the latest onset was "[three] weeks, [three] months." Id. at 4 tbl.2.

2. Petitioner's Expert, Dr. Omid Akbari, Ph.D.[44]

a. Background and Qualifications

Dr. Akbari is a professor of immunology and professor of medicine at the University of Southern California, Keck School of Medicine. Pet. Ex. 45 at 2; Pet. Ex. 46 at 1. He received a Ph.D. in cellular and molecular immunology at the National Institute for Medical Research in London, United Kingdom. Pet. Ex. 46 at 1. Thereafter, he completed a postdoctoral fellowship at Stanford University. Id. Dr. Akbari's research focuses on the "the role of immune tolerance and how immune cells induce autoimmune and allergic diseases." Pet. Ex. 45 at 2. His laboratory research includes multiple studies regarding how an "antigen, allergen, or vaccine can result in an appropriate or dysregulated immune response." Id. at 2-3. Dr. Akbari serves as an associate editor and reviewer on several journals. Id. at 2; Pet. Ex. 46 at 5. He has authored or co-authored numerous publications. Pet. Ex. 45 at 2; Pet. Ex. 46 at 9-16.

[44] Petitioner submitted three expert reports from Dr. Akbari. Pet. Exs. 45, 110, 141. The undersigned does not summarize all of Dr. Akbari's opinions for the sake of brevity and relevance as Dr. Akbari's expert reports are lengthy and include foundational information and opinions not directly relevant to the issues herein. For example, this Decision does not summarize Dr. Akbari's overview of the innate and adaptive immune system and vaccinations. Pet. Ex. 45 at 7-9.

Dr. Akbari is not a medical doctor and is therefore not qualified to diagnose or treat neurological conditions.

b. Opinion

i. Diagnosis

Dr. Akbari's summary of the medical records noted Petitioner was "diagnosed with inflammatory demyelination and TM approximately [three to four] weeks after the administration of the [flu] vaccine and the neuroinflammatory disease and TM eventually progressed to optic neuritis." Pet. Ex. 45 at 6. Dr. Akbari did not offer any opinion about whether these diagnoses were appropriate. He limited his opinions to immunology and causation.

ii. Althen Prong One[45]

Dr. Akbari opined, based on a preponderance of the scientific evidence, that molecular mimicry and induction of inflammasomes are "credible medical theories casually linking the flu vaccination" to Petitioner's demyelinating injury and TM. Pet. Ex. 110 at 23.

Putting forward his molecular mimicry theory, Dr. Akbari explained that the flu vaccine was capable of "stimulating autoreactive T cells." Pet. Ex. 45 at 9. He asserted the components of the flu vaccine Petitioner received contained flu virus peptides able to trigger or polarize autoreactive T cells to myelin proteins and complex sugars associated with gangliosides. Id. at 10. He opined "[t]he immune response elicited to gangliosides mainly targets the sugars on the vaccine, which are also a component of the myelin sheath." Id.

He cited two studies in support of this opinion. Pet. Ex. 45 at 10. Markovic-Plese et al.[46] proposed cross-reactivity of a CD4+ T-cell clone specific for the immunodominant flu virus hemagglutinin peptide (sequence yvkqatlkl) derived from a patient with demyelinating disease including MS. Id. (citing Pet. Ex. 60 at 1). Dr. Akbari explained that in Wucherpfenning and Strominger,[47] a panel of 129 peptides that matched the molecular mimicry motif was tested on seven specific T cell clones from patients with MS. Id. (citing Pet. Ex. 61 at 1). Dr. Akbari asserted that the study found "[s]even viral and one bacterial peptide efficiently activated three of these clones." Id. Notably, however, this study did not include a peptide with the "yvkqstlkl"

[45] For the sake of brevity and clarity, this Decision only briefly covers the two major theories from his reports, molecular mimicry and induction of inflammasomes.

[46] Silva Markovic-Plese et al., High Level of Cross-Reactivity in Influenza Virus Hemagglutinin-Specific CD4+ T-Cell Response: Implications for the Initiation of Autoimmune Response in Multiple Sclerosis, 169 J. Neuroimmunology 31 (2005).

[47] Kai W. Wucherpfennig & Jack L. Strominger, Molecular Mimicry in T Cell-Mediated Autoimmunity: Viral Peptides Activate Human T Cell Clones Specific for Myelin Basic Protein, 80 Cell 695 (1995).

motif. See Pet. Ex. 61; see also Resp. Ex. C at 3 (noting that the study by Wucherpfenning and Strominger "does not contain a peptide with the 'yvkqstlkl' motif").

Next, Dr. Akbari proposed homology between the major protein in the myelin sheath, MBP, with flu A strain in the flu vaccine. Pet. Ex. 45 at 10. He opined the amino acid sequence of the hemagglutinin in the vaccine, fyknli, has "high homology" with the MBP, ffkniv, with the only variations being a substitution of amino acid phenylalanine ("f") for tyrosine ("y"), and amino acids, leucine ("l") and isoleucine ("i"). Id. He characterized this as "another undeniable example of a molecular mimicry with [flu] A strains that appeared in vaccine that [Petitioner] received." Id.

Dr. Akbari explained that as few "as [five] amino acid homology within 12 amino acids is sufficient to induce signs of local and systemic neurological disorders in animal models." Pet. Ex. 45 at 10 (citing Pet. Exs. 62-63). He cited Gautam et al.[48] for the proposition that homology of just five amino acids with a self-peptide can induce clinical signs experimental autoimmune encephalomyelitis ("EAE").[49] Pet. Ex. 63 at 2; see also Pet. Ex. 62.[50]

According to Dr. Akbari, molecular mimicry can occur "even in the absence of any true sequence or structural homology" because "of the level of degeneracy[51] necessary for the human immune system to recognize such an infinite number of foreign antigens." Pet. Ex. 45 at 11. He further asserted that structural homology may be "just as important as sequence homology." Id. (citing Pet. Ex. 64).[52] Dr. Akbari explained structural homology as the idea that "there are crucial residues (rather than significant portions of amino acid sequence) that are conserved and give rise to the ability of T cells or autoantibodies to cross-react." Id.

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[48] Anand M. Gautam et al., A Viral Peptide with Limited Homology to a Self Peptide Can Induce Clinical Signs of Experimental Autoimmune Encephalomyelitis, 161 J. Immunology 60 (1998).

[49] EAE is "is an autoimmune demyelinating disease induced by immunizing susceptible mouse, rat, or guinea pig strains with myelin basic protein . . . or with encephalitogenic peptide fragments." Resp. Ex. C-4 at 1 (Dawn E. Smilek et al., A Single Amino Acid Change in a Myelin Basic Protein Peptide Confers the Capacity to Prevent Rather Than Induce Experimental Autoimmune Encephalomyelitis, 88 Proc. Nat'l Acad. Sci. U.S. Am. 9633 (1991)).

[50] K.W. Wucherpfennig et al., Recognition of the Immunodominant Myelin Basic Protein Peptide by Autoantibodies and HLA-DR2-Restricted T Cell Clones from Multiple Sclerosis Patients. Identity of Key Contact Residues in the B-Cell and T-Cell Epitopes, 100 J. Clinical Investigation 1114 (1997).

[51] Dr. Akbari stated that "degeneracy occurs when structurally dissimilar components can perform similar functions under certain conditions." Pet. Ex. 45 at 11 n.2.

[52] Adam P. Kohm et al., Mimicking the Way to Autoimmunity: An Evolving Theory of Sequence and Structural Homology, 11 Trends Microbiology 101 (2003).

After his discussion of examples of homology, Dr. Akbari turned to the subject of induction of broadly reactive antibodies referenced in a study published in 2022, by Labombarde et al.,[53] which "prov[ed] molecular mimicry via flu peptides." Pet. Ex. 45 at 12 (citing Pet. Ex. 66). The authors found "induction of broadly reactive [flu] antibodies increases susceptibility to autoimmunity" in the context of "inflammation or genetic susceptibility." Pet. Ex. 66 at 2. Specifically, they reported "that inducing broadly reactive [flu] antibodies increases autoreactive antibodies in humans and mice and exacerbates disease in four distinct models of autoimmune disease." Id. The authors induced models of SLE, MS, and GBS in mice. Id. The authors concluded, however, that "self-tolerance mechanisms limit the prevalence of broadly reactive [flu] antibodies." Id. Further, in the study, the mice were treated with the immunosuppressant drug rapamycin[54] to lead to more broadly reactive flu antibodies. Id. at 4-6. The authors noted that a limitation of the study was the inability to test "whether elevated levels of broadly reactive antibodies increase the risk of autoimmunity in humans." Id. at 14-15.

The next topic raised by Dr. Akbari was the role of EBV infection in the pathogenesis of demyelinating disease. Pet. Ex. 45 at 14. Dr. Akbari acknowledged that EBV is the "leading cause of autoreactive immune cells capable of causing demyelinating disease via molecular mimicry." Id. In support, he cited studies by Bjornevik et al.[55] and Lanz et al.[56] Pet. Exs. 67-68.

In Bjornevik et al., the authors conducted a cohort study of 10 million young adults in the U.S. military and identified 995 patients who were diagnosed with the demyelinating disease

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[53] Jocelyn Labombarde et al., Induction of Broadly Reactive Influenza Antibodies Increases Susceptibility to Autoimmunity, 38 Cell Reps. 110482 (2022).

[54] Rapamycin, also known as sirolimus, is an "a macrolide antibiotic obtained from a variant of *Streptomyces hygroscopicus*, having immunosuppressant properties." Sirolimus, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=45959 (last visited May 12, 2025); see also Resp. Ex. F-3 (Lilin Ye et al., mTOR Promotes Antiviral Humoral Immunity by Differentially Regulating CD4 Helper T Cell and B Cell Responses, 91 J. Virology e01653 (2017)) (discussing of the use of rapamycin to "stimulate vaccine-induced immunity" in animal models).

[55] Kjetil Bjornevik et al., Longitudinal Analysis Reveals High Prevalence of Epstein-Barr Virus Associated with Multiple Sclerosis, 375 Science 269 (2022). In Bjornevik et al., EBV was described as "a human herpesvirus that after infection persists in latent form in B lymphocytes throughout the life of the host." Pet. Ex. 67 at 1. The "causal role of EBV is supported by the increased MS risk after infectious mononucleosis, elevated serum antibody titers against EBV nuclear antigens (EBNAs), and by the presence of EBV in MS demyelinated lesions reported in some, but not all, pathological studies." Id. (internal citations omitted). The authors concluded that the "findings cannot be explained by any known risk factor for MS and suggest EBV as the leading cause of MS." Id.

[56] Toblas V. Lanz et al., Clonally Expanded B Cells in Multiple Sclerosis Bind EBV EBNA1 and GlialCAM, 603 Nature 321 (2022).

MS. Pet. Ex. 67 at 1. "Risk of MS increased 32-fold after infection with EBV but was not increased after infection with other viruses." Id. In Lanz et al., the authors explored the mechanistic link between EBV and MS using animal models and demonstrated a "high-affinity molecular mimicry" between the EBV transcription factor EBV nuclear antigen 1 ("EBNA1") and the CNS protein glial cell adhesion molecule ("GlialCAM"). Pet. Ex. 68 at 1. The study showed the "functional evidence" of this homology and described how it caused MS. Id. A third paper, by Bar-Or et al.,[57] reviewed the studies by Bjornevick et al. and Lanz et al. and concluded that they provided "robust epidemiological" evidence that EBV probably plays a "requisite role for the development of MS of most patients." Pet. Ex. 69 at 2. Of note, these papers did not mention the flu virus or flu vaccine. Nor did they describe any role that either the flu virus or vaccine played in the pathogenesis of MS or the mechanistic link between EBV and MS.

Citing these studies, Dr. Akbari suggested that EBV specific lymphocytes, "in the form of memory B and T cells," could contribute to "cross-reactivity of T cells that recognize[d] EBV and [flu] A virus," especially in patients who are HLA-A2+. Pet. Ex. 45 at 15. He also cited a paper by Clute et al., that purported to show that EBV can "cross-react with [flu] A and vice versa" to assert that "these clones . . . are capable of causing demyelination and TM." Id. However, his assertion that EBV can cross react with flu A does not appear to be based on findings described by Bjornevik et al. or Lanz et al. Further, the paper by Clute et al. was not filed into the record.

In his second report, Dr. Akbari acknowledged that homology alone is not sufficient to cause autoimmunity via molecular mimicry. Pet. Ex. 110 at 12. He opined that "a second (or possibly third or multiple) signal[s]" are often essential to induce disease Id. at 22. These include loss of tolerance and/or immune homeostasis. Id. He opined that the flu vaccine could stimulate regulatory T cells ("Tregs") or T effector cells ("Teff cells") to cause imbalances between these cell types which normally maintain homeostasis. Pet. Ex. 45 at 16-17; see also Pet. Ex. 110 at 14. For example, he opined that a decrease in the levels of Tregs and Teff cells can result in "hyper-activation" which contributes to autoimmune illnesses such as neuropathy and TM.[58] Pet. Ex. 45 at 16-17.

Turning to his next major theory, Dr. Akbari identified induction of inflammasomes as a causal mechanism linking flu vaccine and demyelinating injuries. Although Dr. Akbari identified inflammasomes as a relevant mechanism, his discussion about this mechanism was confusing and it was difficult to discern the relevance to this case. Relying first on Crooke et

[57] Amit Bar-Or et al., Guilty by Association: Epstein-Barr Virus in Multiple Sclerosis, 28 Nature Med. 900 (2022).

[58] For Dr. Akbari's discussion of Treg and Teff cells, see Pet. Ex. 45 at 16-17; Pet. Ex. 110 at 12-14.

al.,[59] Dr. Akbari asserted that the flu vaccine is capable of inducing inflammasomes[60] in humans. Pet. Ex. 110 at 7 (citing Pet. Ex. 117); Pet. Ex. 45 at 21 (citing Pet. Ex. 90). Crooke et al. studied the blood tests of 147 adults separated into young (aged 18-39) and old (aged 65-92) groups, pre- and post-flu vaccination. Pet. Ex. 90 at 1. Post-vaccination testing was done at 24 hours and 28 days. Id. at 2. The study showed no significant age-related differences in inflammasome activity in macrophages. Id. at 9. It is not clear how the article is relevant here.[61]

In addition to inflammasomes, Dr. Akbari discussed and cited papers about cytokines.[62] One paper cited by Dr. Akbari, authored by Dixit et al.,[63] reported that 59 patients with acute TM had elevated levels of cytokines in their CSF. Pet. Ex. 130 at 1. Two cytokines (IL-6 and IL-8) were "significantly associated" with disease severity. Id. The authors suggested that the findings may suggest "that the dysregulated production of cytokines contributes [to] the pathogenesis of acute [TM]." Id. at 5-6. The authors summarized the existing information about cytokines relative to MS and NMO, noting that the findings "may have an important bearing in terms of treatment as well as prognosis" of TM and NMO. Id. at 6. The article did not discuss vaccines.

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[59] Stephen N. Crooke et al., Inflammasome Activity in Response to Influenza Vaccination Is Maintained in Monocyte-Derived Peripheral Blood Macrophages in Older Adults, 2 Frontiers Aging 1719103 (2021). Crooke et al. was filed as both Petitioner's Exhibit 90 and 117.

[60] Inflammasomes are "a complex of cryopyrin, caspase-1, and other proteins, found in phagocytic cells and related to the body's system of innate immunity. Assembly of the inflammasome leads to activation of caspase-1 and resultant cleavage and activation of interleukins IL-1β and IL18 in the inflammatory response." Inflammasome, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=25203 (last visited May 12, 2025).

[61] For further support, Dr. Akbari discussed and cited to additional studies about inflammasomes and proinflammatory cytokines. See Pet. Exs. 118-129. One the whole, these articles do not seem to directly relate to the question of how the flu vaccine can cause a CNS demyelinating illness. The undersigned does not summarize most of Dr. Akbari's opinions about inflammasomes and cytokines as these opinions appear less relevant than his discussion of molecular mimicry and the adaptive immune response. For the specific portions of his reports related to inflammasomes and cytokines, see Pet. Ex. 45 at 20-22; Pet. Ex. 110 at 7-11.

[62] Cytokine is "a generic term for nonantibody proteins released by one cell population (e.g., primed T lymphocytes) on contact with a specific antigen, which act as intercellular mediators, as in the generation of an immune response. Examples include lymphokines and monokines." Cytokine, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=12428 (last visited May 14, 2025).

[63] Puneet Dixit et al., Cytokines and Matrix Metalloproteinases in the Cerebrospinal Fluid of Patients with Acute Transverse Myelitis: An Outcome Analysis, 65 Inflammation Rsch. 125 (2016).

In support of vaccine causation of TM, Dr. Akbari cited Agmon-Levin et al., a study of case reports published between 1970 and 2009. Pet. Ex. 81 at 2. The authors identified 37 cases of post-vaccination TM; two cases followed a flu vaccine. Id. at 2, 3 tbl.1. While noting a "possible" association between vaccination and TM, the authors stated that "up to 40% of TM cases are associated with a preceding infectious illness. . . . Different infectious agents have been implicated in the pathogenesis of TM," including EBV. Id. at 2. "In most cases symptoms of TM begin after the patient had recovered from the infectious disease. . . . TM appears not to be a direct infectious process, but rather an autoimmune response triggered by the infectious agents." Id.

In his second and third reports, Dr. Akbari opined at length that Baxter et al.[64] was unreliable due to the authors' conflicts of interests which "inherently introduces a potential bias into the research." Pet. Ex. 141 at 2; see also Pet. Ex. 110 at 1-7; Resp. Ex. A-6. Dr. Akbari discusses conflicts of interest, including financial conflicts, and he addresses concerns about data derived from the Vaccine Safety Database ("VSD").[65] See Pet. Ex. 110 at 1-7; Pet. Ex. 141 at 3-6.

iii. Althen Prongs Two and Three

Dr. Akbari opined "to high degree of certainty, and by a preponderance of scientific evidence, had it not been for the flu vaccination," Petitioner "would not have developed [an] inflammatory demyelinating disease." Pet. Ex. 45 at 27. He agreed with Dr. Ghacibeh that "there is a clear logical sequence of events supporting a cause and effect relationship between exposure to the vaccine and the development of immune-related illnesses." Id.

Based on his review of the record, Dr. Akbari found the timing of the vaccine and the development of TM approximately three to four weeks later to be consistent with the medical literature and the theories offered. Pet. Ex. 45 at 26-27. He concluded that a "proximate temporal relationship" existed between receipt of the flu vaccine and Petitioner's injury. Id. at 27.

3. Respondent's Expert, Dr. Mark B. Bromberg, M.D., Ph.D.[66]

a. Background and Qualifications

Dr. Bromberg is a professor of neurology at the University of Utah. Resp. Ex. A at 1. He is board certified in neurology and clinical neurophysiology by the American Board of

[64] Rodger Baxter et al., Acute Demyelinating Events Following Vaccines: A Case-Centered Analysis, 63 Clinical Infectious Diseases 1546 (2016). This article was also filed as Pet. Ex. 91.

[65] The undersigned's Decision in this matter does not turn on the study by Baxter et al. Due to relevance and the sake of brevity, the undersigned acknowledges Dr. Akbari's opinions about Baxter et al. but does not summarize them herein.

[66] Respondent submitted two expert reports from Dr. Bromberg. Resp. Exs. A, D.

Psychiatry and Neurology and he also holds a certification from the American Board of Electrodiagnostic Medicine. Id.; Resp. Ex. A-1 at 6. He completed his M.D., his residency, and a neuromuscular fellowship at the University of Michigan. Resp. Ex. A-1 at 1. Additionally, Dr. Bromberg holds a Ph.D. in Neurophysiology from the University of Vermont. Id. Dr. Bromberg completed a postdoctoral fellowship at the University of Washington. Id. His research focus is on the peripheral nervous system. Resp. Ex. A at 1. Dr. Bromberg sees patients in a general neurology clinic. Id. In his clinical practice, he has "diagnosed cases of [TM] and compressive myelopathies." Id. Dr. Bromberg serves as an associate editor and reviewer for several journals. Resp. Ex. A-1 at 2-3. He has published extensively on neuromuscular disorders and peripheral nerve disorders. Resp. Ex. A at 1; Resp. Ex. A-1 at 19-43.

b. Opinion

Dr. Bromberg opined that, more likely than not, Petitioner did not have TM. Resp. Ex. A at 13; Resp. Ex. D at 2. He further opined that Petitioner's "constellation of symptoms was probably not caused by her [flu] vaccine." Resp. Ex. A at 13.

i. Diagnosis

Dr. Bromberg opined that Petitioner's symptoms were "not sufficient to make a diagnosis of [TM]." Resp. Ex. A at 13.

Dr. Bromberg explained that Petitioner's clinical records did not provide evidence that she met the diagnostic criteria for acute TM.[67] Resp. Ex. A at 10 (citing Resp. Ex. A-2). The diagnostic criteria require "pathology localized to the spinal cord," "bilateral symptoms/signs," "clearly defined sensory level," "CSF pleocytosis or increased IgG synthesis or gadolinium enhancement" on MRI, and "progression to nadir [four] hours to 21 days after symptoms onset." Id. at 10-11.

Applying this criteria, Dr. Bromberg opined that Petitioner's MRI findings were "not consistent with pathologic damages to nerves in the spinal cord." Resp. Ex. A at 11. He opined that Petitioner's MRI showed a spinal cord lesion limited to a single level with no enhancement. Resp. Ex. D at 1. Thus, Dr. Bromberg questioned the clinical significance of the limited findings seen on Petitioner's thoracic MRI. Resp. Ex. A at 11. In support, he noted that Abbatemarco et al. reported 24.3% of patients with TM had MRI abnormalities spanning three or more spinal cord segments constituting longitudinally extensive TM. Id. (citing Pet. Ex. 44 at 4). He agreed with Dr. Ghacibeh that Abbatemarco et al. also noted that more than 70% of TM patients had "spinal cord lesions spanning less than three segments," however, Dr. Bromberg noted that no data was provided about the number of patients who had lesions "limited to a single level." Resp. Ex. D at 1.

[67] These criteria were proposed by the TM Consortium Working Group and published in 2002. See Resp. Ex. A-2. The TM Consortium Working Group members included physicians from a wide range of neurology departments worldwide, as set forth in the appendix. Id. at 4, 7.

Dr. Bromberg briefly described the spinal cord. Resp. Ex. A at 10. It is 1.0 to 1.5 cm in diameter, with its outermost part made of "descending corticomotor axons and ascending sensory axons." Id. The "middle region" contains "motor axons projecting to muscles and interneurons connecting axons from peripheral nerves to motor nerves and other short axons." Id. The segments of the spinal cord are referenced based on the adjacent numbered vertebrae. Id. When the thoracic segments of the spinal cord are injured, impulses to descending motor axons affecting leg muscles are affected as well as "ascending sensory axons carrying information upward." Id. Lesions in the spinal cord "can involve both right and left sides of the cords or a single side." Id.

Next, Dr. Bromberg noted that Petitioner's lesion was limited to the central spinal cord. Resp. Ex. A at 11. He explained that central cord involvement implicates involvement of corticospinal fibers which impact spasticity and abnormally reflexes which were not found during Petitioner's examination. Id. Responding to Dr. Ghacibeh's opinion that Petitioner's thoracic lesion could "possibly only involve sensory fibers," Dr. Bromberg explained that Petitioner's MRI imaging report was "not precise" and did not describe the location or extent of cord involvement. Resp. Ex. D at 1. He maintained that the lack of motor symptoms, such as hyperreflexia, was "striking" and argued against a TM diagnosis. Id.

Addressing bilateral signs and symptoms, Dr. Bromberg acknowledged that Petitioner's sensory symptoms were in a bilateral distribution. Resp. Ex. A at 11. But her symptoms were "restricted to sensory disturbances" and she had no motor dysfunction. Id. Dr. Bromberg opined that Petitioner had "no clear or consistent leg weakness." Id. Petitioner's "mild leg weakness" varied between assessments and Dr. Bromberg asserted that such "variability likely represented differing effort in the setting of an expectation of finding." Id. He also noted Petitioner's mild gait abnormality was "intermixed with descriptions of normal gait." Id. Therefore, he concluded that Petitioner had "no objective signs of sensory or motor dysfunction." Id.

Dr. Bromberg opined that Petitioner partially fulfilled the criterion of clearly defined sensory level. Resp. Ex. A at 11. Examinations by treating neurologists "sometimes but not consistently" documented sensory level of "about thoracic level 10." Id. at 9. He reiterated that Petitioner's symptoms were variable and suggested that the stocking distribution of Petitioner's sensory symptoms was more consistent with a peripheral nerve distribution or polyneuropathy, which led her treating neurologists to "suspect" GBS or CIDP. Id. at 11.

Next, Dr. Bromberg argued that Petitioner did not have diagnostic evidence of inflammation. Resp. Ex. A at 9. Her CSF proteins were in the low to normal range on two occasions and there was no evidence of increased IgG synthesis. Id. He also noted that was no gadolinium enhancement on her three MRI studies to suggest inflammation and demyelination. Id.

Finally, Dr. Bromberg opined that Petitioner's symptoms did not have "a clear nadir." Resp. Ex. A at 11. Rather, she had an initial progression of symptoms followed by frequent changes of lessening or worsening symptoms. Id. Additionally, she had improvement in response to IVIG and IV steroids, followed by relapse. Id. She had no sustained response to treatment. Id. Dr. Bromberg opined that this did not fit the clinical course expected for TM. Id.

Dr. Bromberg felt that if Petitioner "truly had clear symptoms of TM," these symptoms would have been noted by her treating neurologists during her early diagnoses and treatments. Resp. Ex. D at 1-2. Dr. Bromberg disagreed with any suggestion that the inconsistency of Petitioner's motor findings were due to a lack of thoroughness or completeness by her treating neurologists. Id. He noted that a "clinical sensory examination is totally subjective, and [Petitioner's] reports were highly variable." Id. at 1. These fluctuations over time, he noted, was "unusual for TM." Id. at 2.

Also in his first report, Dr. Bromberg disagreed that Petitioner had "bladder function abnormalities" consistent with TM. Resp. Ex. A at 12. However, in his second report, he acknowledged that Petitioner had symptoms of urinary urgency beginning approximately eight months after vaccination. Resp. Ex. D at 2. He opined that such a delay was inconsistent with TM, which is characterized by an acute onset. Id. Further, he explained Petitioner's diagnostic tests demonstrated complete bladder emptying, which he opined did not suggest that she had a neurogenic bladder as would be expected in TM. Id.

Regarding optic neuritis, in his first report, Dr. Bromberg opined that Petitioner did not have "an episode consistent with optic neuritis." Resp. Ex. A at 12. He acknowledged that Petitioner was initially diagnosed with optic neuritis by Dr. Mack but he asserted that this diagnosis was "refuted by Dr. Tamhankar." Id. (citing Pet. Ex. 25 at 6). Dr. Bromberg questioned Dr. Mack's optic neuritis diagnosis and argued that he relied on "marginal/questionable" visual field examination with the right eye defect "not corresponding to optic nerve or retinal nerve fiber layer defect" to diagnosis optic neuritis when Petitioner had "an essentially normal exam[ination]." Id. Further, Dr. Bromberg noted that expected inflammation of the optic nerve did not appear on Petitioner's October 2020 MRI. Id. In his subsequent report, Dr. Bromberg deferred to Respondent's neuro-ophthalmologist, Dr. Mackay, regarding Petitioner's optic neuritis diagnosis. Resp. Ex. D at 2.

Regarding other possible diagnoses, Dr. Bromberg opined that the diagnoses of GBS or CIDP were "untenable." Resp. Ex. A at 9. Petitioner's normal conduction study did not support a diagnosis of GBS and Petitioner's lack of motor nerve involvement, repeated normal conductions studies, and normal CSF analysis did not support a diagnosis of CIDP. Id. He further opined that possible diagnoses of NMO and MS were excluded because Petitioner did not have the required symptoms and she had normal laboratory tests. Id. at 10.

ii. Althen Prong One

Dr. Bromberg opined that "any association" between the flu vaccine and TM "is not supported." Resp. Ex. A at 12.

Dr. Bromberg explained that the pathology of TM "is felt to be an immune response to an inciting pathologic event or condition." Resp. Ex. A at 9. The inflammatory pathology "involves B-cells, T-cells[,] and other inflammatory proteins. The consequence is focal damage to myelin cover[ed] axons and to the axons themselves." Id. at 10. Further, inflammatory injury to myelin and axons leads to "a breakdown of the blood-brain vascular barriers" resulting in

"leakage of white cells and protein from blood vessels into the spinal cord tissue and into the CSF." Id. This damage is detected by MRI where "leakage of fluid at the site of the inflammatory lesions changes (enhances) the MRI signal." Id. If contrast is administered for the MRI scan, it may show additional enhancement of the MRI signal. Id.

Regarding vaccination as inciting event, Dr. Bromberg cited to Baxter et al., which found "no statistically significant risk increased risk" of TM in the five-to-28-day risk interval following flu vaccination. Resp. Ex. A at 12 (citing Resp. Ex. A-6 at 5). Baxter et al. performed a case-centered analysis of all cases of TM and ADEM in the VSD population which included data on 18,926,060 doses of flu vaccine given to adults and children between 2007 and 2012. Resp. Ex. A-6 at 1. The authors found "no statistically significant increased risk" of TM post-vaccination "in either the [five]- to 28-day or the [two]- to 42-day risk interval." Id. at 5, 3 tbl.2. The authors further concluded that, for TM, they found "no evidence of a safety concern or an association with subsequent illness. If there is any association, it is <1 per million doses for vaccines other than live zoster and live attenuated [flu] vaccines, and <2 per million doses of these [two] vaccines." Id. at 6.

Dr. Bromberg did not address Dr. Ghacibeh's or Dr. Akbari's theories of causation.

iii. Althen Prong Two

Regarding Althen prong two, Dr. Bromberg argued alternative etiologies, including EBV and exposure to nitrous oxide, existed for Petitioner's condition. Resp. Ex. A at 9, 12; see also Resp. Ex. D at 2. Specifically, Dr. Bromberg opined that EBV was a "possible cause of [Petitioner's] symptoms." Resp. Ex. A at 12.

In support, he noted her treating physician attributed Petitioner's diagnosis—initially GBS and later TM—to EBV. Resp. Ex. A at 9 (citing Pet. Ex. 3 at 31); Resp. Ex. D at 2. For example, on December 18, 2018, Dr. Corti noted Petitioner's "presentation [was] concerning for [GBS] due to active EBV." Pet. Ex. 3 at 31. Later, on January 31, 2019, Dr. Corti opined Petitioner had "T7-8 [TM] with whole body rash, most likely from EBV." Id. at 22. Additionally, Dr. Bromberg noted that Petitioner "had several bouts of EBV, and ha[d] antibodies to the virus." Resp. Ex. A at 12.

Dr. Bromberg provided two case reports linking TM and EBV infections. Resp. Ex. A at 12. Caldas et al.[68] described a previously healthy 28-year-old female patient who developed TM associated with an "acute EBV infection." Resp. Ex. A-4 at 1. The patient had antibody testing which indicated acute EBV infection and serologic studies for other viruses were negative. Id. The authors asserted that this was the "first case where EBV serology suggested etiology at the time of diagnosis." Id. The authors acknowledged that the pathogenesis of TM as a neurological complication of EBV infection remained unclear. Id. They recommended that "EBV infection be suspected in young patients with [TM], and that viral serologies be included in the diagnostic

[68] Carlos Caldas et al., Case Report: Transverse Myelitis Associated with Epstein-Barr Infection, 307 Am. J. Med. Scis. 45 (1994)

workup." Id. An earlier paper by Grose and Feorino[69] reported a 20-year-old female patient who developed acute TM. Resp. Ex. A-5 at 1. A serum draw from her fourth week at the hospital had high levels of EBV which "strongly suggest[ed] either a recent infection or activation." Id.

Dr. Bromberg cited to treating physician Dr. Edelman, who noted on December 18, 2018, that it was "unclear whether [Petitioner's symptoms were] secondary to nitrous oxide or some other chemical, autoimmune." Pet. Ex. 10 at 121; see also Resp. Ex. A at 9. However, Dr. Bromberg acknowledged in his report that "most cases of myelopathy due to nitrous oxide involve chronic inhalation" and Petitioner's exposure to nitrous oxide was limited. Resp. Ex. A at 12.

iv. Althen Prong Three

Dr. Bromberg acknowledged a five to 28 day "risk interval" for TM onset following vaccination. Pet. Ex. A at 12 (citing Resp. Ex. A-6 at 5). Baxter et al. determined five to 28 days to be "the most likely" onset interval for a demyelination illness following vaccination. Resp. Ex A-6 at 3. In their study design, the authors also found an onset interval of two to 42 days appropriate. Id.

Dr. Bromberg agreed that Petitioner's sensory symptoms began on December 3, 2018—25 days post-vaccination. Resp. Ex. A at 9. However, Dr. Bromberg disputed these sensory symptoms were associated with TM. Id.

4. Respondent's Expert, Dr. Devin D. Mackay, M.D.[70]

a. Background and Qualifications

Dr. Mackay is an associate professor of neurology, ophthalmology, and clinical neurosurgeon and the director of neuro-ophthalmology at Indiana University School of Medicine. Resp. Ex. B at 1; Resp. Ex. B-1 at 1. He is board certified in neurology by the American Board of Psychiatry and Neurology. Resp. Ex. B at 2; Resp. Ex. B-1 at 2. He received his M.D. from the University of Virginia and subsequently completed a neurology residency at Havard Medical School at Massachusetts General Hospital and Brigham and Women's Hospital. Resp. Ex. B at 1; Resp. Ex. B-1 at 1. He then completed a fellowship in neuro-ophthalmology at Emory University. Resp. Ex. B at 1; Resp. Ex. B-1 at 1. Dr. Mackay maintains an outpatient clinical practice of neuro-ophthalmology as well as inpatient clinical service of general inpatient neurology. Resp. Ex. B at 2. He has treated over 6,300 patients, "including many with demyelinating diseases of the [CNS]." Id. Dr. Mackay serves on one editorial board and has been a reviewer for several journals. Resp. Ex. B-1 at 11. Dr. Mackay

[69] Charles Grose & P.M. Feorino, Epstein-Barr Virus and Transverse Myelitis, 301 Lancet re892 (1973).

[70] Respondent submitted two expert reports from Dr. Mackay. Resp. Exs. B, E.

has lectured on and authored, or co-authored, publications on topics related to neuro-ophthalmology. Id. at 6, 9, 12-17; Resp. Ex. B at 1.

b. Opinion

Dr. Mackay opined, to a reasonable degree of medical certainty, that Petitioner did not suffer optic neuritis or NMO due to the flu vaccine she received on November 8, 2018. Resp. Ex. E at 4; Resp. Ex. B at 12

i. Diagnosis

Dr. Mackay opined that Petitioner was "given multiple diagnoses" by different physicians from the onset of her symptoms in December 2018, including the diagnosis of "optic neuritis is July 2020." Resp. Ex. B at 11. He opined, however, that Petitioner's diagnosis of optic neuritis was not supported by her clinical history, physical examination findings, or neuroimaging studies. Id.

According to Dr. Mackay, optic neuritis is "an inflammatory attack of the optic nerve . . . caused by inflammatory or infectious diseases." Resp. Ex. B at 11. It is "a relatively common cause of vision loss in adults, affecting [one] to [five] individuals per 100,000 per year." Resp. Ex. B-2 at 2.[71] Optic neuritis may be associated with a demyelinating disease, like MS or NMOSD.[72] Id. And it is a "common clinical manifestation of [CNS] inflammation." Resp. Ex. E-1 at 1. There are multiple causes of the condition, including autoimmunity, infection, and demyelination, among others. Id. Optic neuritis presents as "acute, unilateral, painful vision loss." Id. at 2. Physical examination usually "reveals visual acuity loss, visual field loss, color vision deficits, and an [APD] in the affected eye." Id.

Dr. Mackay explained that Petitioner's July 2020 optic neuritis diagnosis by Dr. Mack was "based on subjective blurry vision in the right eye." Resp. Ex. B at 11. However, Petitioner's visual acuity was "nearly normal" with 20/30 in the right eye and she had no APD. Id. Dr. Mack acknowledged it was "odd" that Petitioner did not have any ADP. Resp. Ex. E at 3 (quoting Pet. Ex. 7 at 10).

According to Dr. Mackay, the lack of APD in the July 2020 examination refuted the diagnosis of optic neuritis because the presence of ADP is "a fundamental physical examination finding" for any optic nerve dysfunction, including transient optic neuritis. Resp. Ex. E at 3 (citing Resp. Ex. E-1). Bennet explained "the absence of [ADP] should always raise diagnostic concerns unless the patient has bilateral involvement or a history of optic neuropathy in the fellow eye." Resp. Ex. E-1 at 2.

[71] Sarah Chaoying Xu et al., Optical Coherence Tomography Is Highly Sensitive in Detecting Prior Optic Neuritis, 92 Neurology 527 (2019).

[72] For the diagnostic criteria specific to NMOSD, see Resp. Ex. B-3.

Further, later optical examination and testing performed by Dr. Tamhankar on October 6, 2020 revealed normal results. Resp. Ex. B at 11; Resp. Ex. E at 3. Dr. Mackay explained that Petitioner had "visual acuity of 20/20 in both eyes, full color vision, and normal optic nerve appearance" at her October 6, 2020 visit. Resp. Ex. B at 11. Additionally, Dr. Mackay noted that as a neuro-ophthalmologist, Dr. Tamhankar was "a specialist in optic neuritis." Id. Dr. Mackay agreed with Dr. Tamhankar's statement that it would be unusual for optic neuritis to occur during an IVIG taper, which Petitioner was undergoing at the onset of her optical symptoms. Id. at 12; Resp. Ex. E at 3.

Moreover, Dr. Mackay opined Petitioner's OCT on October 6, 2020 was normal and showed "no difference in retinal nerve fiber layer thickness or ganglion cell layer thickness" in her eyes. Resp. Ex. B at 11. There was "no evidence of even slight ganglion cell damage." Id. at 11-12; Resp. Ex. E at 3. A study by Xu at al. found ganglion cell layer thickness, detected based on asymmetry, to be a highly sensitive measure for detecting prior optic neuritis. Resp. Ex. B-2 at 4. The asymmetry existed even in patients with high rates of visual recovery with normal visual acuity, visual fields, and color visions at the time of the OCT. Id.

Dr. Mackay asserted that Petitioner's symmetrical OCT results refute Dr. Ghacibeh's opinion that Petitioner experienced a "mild and fully reversible" episode of optic neuritis. Resp. Ex. E at 3. Dr. Mackay further noted that "misdiagnosis of optic neuritis is not uncommon." Id. A study by Stunkel et al.,[73] which reviewed 496 neuro-ophthalmology visits, found that only 11 out of 16 patients referred with a diagnosis of optic neuritis were given a final diagnosis of optic neuritis. Resp. Ex. E-2 at 3 tbl.2.

Finally, Dr. Mackay noted that Petitioner's description of her symptoms in July 2020, including blurry and double vision, headache and nausea, right eye tearing, and the right eye feeling like it is "catching when trying to look left," could be attributed to many different conditions. Resp. Ex. E at 4 (quoting Pet. Ex. 7 at 8). He also noted the symptoms of double vision, tearing, or feeling like the eye is "catching" are not caused by optic neuritis. Id. Since he did not personally examine Petitioner, Dr. Mackay declined to provide an alternative diagnosis for Petitioner's symptoms based solely on the records he reviewed. Id.

Addressing NMO, Dr. Mackay disagreed with Dr. Ghacibeh's opinion that NMO was a plausible diagnosis. Resp. Ex. B at 12. NMO is an inflammatory autoimmune condition involving both the optic nerve and spinal cord. Resp. Ex. B-3 at 1. It is "an inflammatory CNS syndrome distinct from MS associated with serum [AQP4-IgG]." Id. Although NMO was previously thought to be a monophasic disorder requiring simultaneous optic nerve and spinal cord involvement, newer terminology and criteria reference the presence (or absence) of AQP4-IgG serology, which are "highly specific" for NMO. Id.

[73] Leanne Stunkel et al., Patient Harm Due to Diagnostic Error of Neuro-Ophthalmologic Conditions, 128 Ophthalmology 1356 (2021).

Dr. Mackay explained that Petitioner did not meet the diagnostic criteria for NMO.[74] Resp. Ex. B at 12. First, Petitioner did not have AQP4 antibodies. Id. Therefore, to meet the diagnostic criteria, Petitioner needed to have optic neuritis, longitudinally extensive TM, or area postrema syndrome. Id. (citing Resp. Ex. B-3 at 3). As described above, Dr. Mackay opined that Petitioner did not have optic neuritis. Id. Next, Petitioner's "alleged" TM "spanned, at most, two segments of the spinal cord" while longitudinally extensive TM must span three contiguous segments of the spinal cord. Id. Thus, she did not have longitudinally extensive TM. Id. Dr. Mackay further asserted that "even 'atypical' NMO" presentations must meet diagnostic criteria. Id. Thus, Dr. Mackay disagreed that NMO was a plausible diagnosis. Id.

Dr. Mackay acknowledged that Petitioner had "multiple neurologic diagnoses from different clinicians from her symptoms that began in December 2018." Resp. Ex. B at 11. He did not provide a specific opinion about whether a diagnosis of TM was appropriate.

ii. Causation

Dr. Mackay opined, to a reasonable degree of medical certainty, that Petitioner "did not suffer optic neuritis or any vision damage due to the [flu] vaccine." Resp. Ex. E at 4; Resp. Ex. B at 13.

Dr. Mackay asserted that the medical literature provided by Dr. Ghacibeh did not support an association between vaccination and NMO. Resp. Ex. B at 12-13. First, he noted that Petitioner's clinical presentation and the cases presented in the medical literature "were quite different." Id. at 12. For example, in Ismail and Salama, three cases of "NMO-like presentation were identified" following COVID-19 vaccine. Pet. Ex. 37 at 2. Two of the cases involved longitudinally extensive TM and the other involved AQP4 antibodies. Id. at 12-13. Another study by Karussis and Petrou identified seven cases of NMO following vaccination with each of the seven demonstrating longitudinally extensive TM. Pet. Ex. 31 at 1. In Karussis and Petrou, most cases of NMO followed HPV vaccine which "raise[d] the possibility of cross-reactively between the used viral antigens and [AQP4]." Resp. Ex. B at 12 (citing Pet. Ex. 31). Dr. Mackay noted that Petitioner had neither longitudinally extensive TM nor AQP4 antibodies. Id.

Dr. Mackay critiqued two other studies referenced by Dr. Ghacibeh. Resp. Ex. B at 12. He noted that in Langer-Gould et al., which identified 780 cases of CNS demyelinating cases following vaccination, the authors did not identify the numbers of optic neuritis or NMO cases. Id. (citing Pet. Ex. 35). While Poser identified cases of spinal cord and optic nerve involvement, Poser did not identify any cases of NMO, did not specify "the length of spinal cord involvement," and did not identify any cases involved longitudinally extensive TM. Id. (citing Pet. Ex. 32).

Lastly, Dr. Mackay disagreed with Dr. Ghacibeh's assertion that differences between the clinical presentations in the medical literature and the clinical presentation of Petitioner provided further support that Petitioner's condition was caused by vaccination. Resp. Ex. E at 3; see also Pet. Ex. 43 at 4. Dr. Mackay argued "that vaccinations may be capable of provoking a range of

[74] For the diagnostic criteria, see Resp. Ex. B-3 at 3 tbl.1.

different clinical presentations in different patients does not provide license to conclude that any symptoms experienced by any patient at any point following a vaccination must be due to the vaccination." Resp. Ex. E at 3.

5. Respondent's Expert, Dr. William F. Hawse, Ph.D.[75]

a. Background and Qualifications

Dr. Hawse is an assistant professor in the department of immunology at the University of Pittsburgh School of Medicine. Resp. Ex. C at 1; Resp. Ex. C-1 at 1. He received a Ph.D. in biophysical chemistry from the Johns Hopkins School of Medicine. Resp. Ex. C-1 at 1. Thereafter, he completed postdoctoral fellowships at the University of Notre Dame and the University of Pittsburgh. Id. His background is in "in basic immunological mechanisms focused on adaptive immune responses." Resp. Ex. C at 1. Dr. Hawse's research focuses on $CD4^+$ T cell differentiation and immune tolerance as well as "identifying therapeutic strategies for autoimmune diseases" and he has worked with research groups studying B cell differentiation. Id. Dr. Hawse has authored, and co-authored, journal articles on these topics. Id.; Resp. Ex. C-1 at 2-4.

Dr. Hawse is not a medical doctor and is not qualified to diagnose or treat neurological conditions.

b. Opinion

Dr. Hawse opined that, more likely than not, vaccination did not cause Petitioner's injury. Resp. Ex. C at 10; Resp. Ex. F at 13.

i. Diagnosis

Dr. Hawse did not offer any opinion about diagnosis or the clinical aspects of Petitioner's case. Resp. Ex. C at 2. Instead, he limited his opinions to immunology and causation. Id.

ii. Althen Prong One

Dr. Hawse summarized and responded to Dr. Akbari's proposed causation mechanisms. Resp. Ex. C at 9. He did not "endorse Dr. Akbari's theories" and instead opined "that, more likely than not, vaccination did not cause [P]etitioner's injury." Resp. Ex. C at 9-10.

Addressing molecular mimicry, Dr. Hawse opined that the specific molecular mimics identified by Dr. Akbari were "unlikely to cause . . . [P]etitioner's disease." Resp. Ex. F at 12; see also Resp. Ex. C at 9. First, Dr. Hawse opined that the proposed peptides are not processed or presented as antigens in humans. Resp. Ex. C at 9. Dr. Akbari identified "yvkqstlkl" as a component of flu hemagglutinin protein that shared homology with components of the CNS. Id. at 2 (citing Pet. Ex. 45 at 10). However, Dr. Hawse explained that it was unlikely that that

[75] Respondent submitted two expert reports from Dr. Hawse. Resp. Exs. C, F.

humans generate this specific peptide fragment. Id. In support, he explained that Cassotta et al.[76] found that a peptide with the "yvkqstlkl" sequence was "not naturally processed or presented by human cells." Id. at 2-3 (citing Resp. Ex. C-2 at tbl.S6).[77] Additionally, "T cells isolated in humans immunized with the flu vaccine did not respond to a peptide containing 'yvkqstlkl.'" Id. at 3 (citing Resp. Ex. C-2). Dr. Hawse concluded that it was "unlikely" that the flu vaccine would cause injury through this cross-reaction mechanism when individuals receiving the flu vaccine were unlikely to generate either an antigen containing "yvkqstlkl" or have a "CD4+ T cell immune response towards a peptide containing 'yvkqstlkl.'" Id.

Addressing the proposed homology between amino acid sequence "fyknli" in flu hemagglutinin and the sequence of "ffkniv" in MBP, Dr. Hawse opined that it was "improbable" that an antigen with those three amino acid substitutions would trigger a cross-reactive response. Resp. Ex. C at 3-4. In support, Dr. Hawse cited a study by Anderton et al.[78] that "systematically mutated amino acids of the 'ffkniv' region of [MBP]" to study the impact on T cell activation. Id. at 3 (citing Resp. Ex. C-3). The authors found that single point mutation to amino acid position 92, 95, and 96 in MBP prevented T cell activation. Id. (citing Resp. Ex. C-3 at 3 fig.4).

Dr. Hawse also disagreed that homology of as few as five amino acids within 12 amino acids, or a 42% identity, could induce an autoimmune response. Resp. Ex. C at 3. He asserted that the idea of 42% identity is not reliable given that that even a single amino acid substitution can "completely block an autoimmune response." Id. For example, Smilek at al. reported that that the substitution of a single amino acid in MBP peptide prevented rather than induced EAE in animal models.[79] Resp. Ex. C-4 at 4. Likewise, Nicholson et al.[80] found single amino acid substitution to myelin proteolipid protein inhibited the development of EAE in animal models. Resp. Ex. C at 5 (citing Resp. Ex. C-5 at 1); see also Resp. Ex. C-6 at 1 (noting a single amino acid substitution to a peptide antigen can inhibit T cell activation).[81] Moreover, Dr. Hawse

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[76] Antonio Cassotta et al., Deciphering and Predicting $CD4^+$ T Cell Immunodominance of Influenza Virus Hemagglutinin, 217 J. Experimental Med. e20200206 (2020).

[77] Table S6 was not included in the printed version of the article but was published online as a supplemental table and may be found at https://doi.org/10.1084/jem.20200206. See Resp. Ex. C-2 at 20. The peptide sequence "yvkqstlkl" does not appear in supplemental table 6.

[78] Stephen M. Anderton et al., Influence of a Dominant Cryptic Epitope on Autoimmune T Cell Tolerance, 3 Nature Immunology 175 (2002)

[79] The peptide Ac1-11 (sequence "asq**k**rpsqrhg") induced EAE, while peptide Ac1-11[4A] (sequence "asq**a**rpsqrhg") prevented EAE. Resp. Ex. C-4 at 4; see also Resp. Ex. C at 4-5

[80] Lindsay B. Nicholson et al., An Altered Peptide Ligand Mediates Immune Deviation and Prevents Autoimmune Encephalomyelitis, 3 Immunity 397 (1995).

[81] Gilbert J. Kersh et al., Structural and Functional Consequences of Altering a Peptide MHC Anchor Residue, 166 J. Immunology 3345 (2001).

argued these studies demonstrate that it is "impossible to predict, using amino acids sequence alone, that a peptide can elicit an autoimmune response." Resp. Ex. C at 6.

Additionally, Dr. Hawse opined that the study by Karin et al.[82] provided further evidence against the idea that a five out of 12 amino acid identity could induce autoimmunity. Resp. Ex. C at 6 (citing Resp. Ex. C-7). Dr. Hawse explained that the authors used a peptide from myelin basic protein containing the sequence "ffkniv" to induce EAE in rats. Id. at 6. Peptides based on this sequence were mutated at amino acid position 87 through 99. Id. Dr. Hawse argued that using the "[five] out of 12 identify rule, all the [altered] peptides should . . . induce EAE." Id. at 6. However, none of the rats who received a peptide with mutations at position 90, 91, 95, or 96 developed EAE. Id. at 6, 6 tbl.1.

Dr. Hawse criticized Dr. Akbari's reliance on Labombarde et al. for the proposition that the flu vaccine "induces broadly reactive antibodies" resulting in increased autoreactive antibodies capable of inducing demyelination. Resp. Ex. F at 11; Resp. Ex. C at 6-7. The Labombarde et al. study utilized rapamycin at the time of vaccination to induce broadly neutralizing antibodies in the animal models. Resp. Ex. F at 11 (citing Pet. Ex. 66 at 4). Dr. Hawse asserted that without rapamycin, broadly neutralizing antibodies "do not readily form" and thus, there is "no increase in autoreactive responses." Id. Further, rapamycin has a "profound impact on immune cells" that must be taken into account when interpreting the results of studies that use rapamycin treatment. Id. at 12 (citing Resp. Ex. F-3). He noted that Petitioner was not receiving rapamycin at the time of her vaccination and opined that without the addition of rapamycin the reaction described in Labombarde et al. would not occur in Petitioner. Id.

Dr. Hawse acknowledged that Bjornevik et al. provided a molecular mimicry mechanism linking EBV and MS. Resp. Ex. C at 7. However, he criticized Dr. Akbari extrapolating the link between EBV and MS to the flu vaccine and demyelinating disease. Id. Further, Dr. Hawse noted that Bjornevik et al. "found flu infection is not associated with MS development." Id. (citing Pet. Ex. 67 at 3). Specifically, the authors compared serum samples from 30 MS cases and 30 matched controls and found the peptide-specific antibody data "support[ed] the specificity of the association between EBV and MS and argues against a second hit from another virus playing a major role in MS etiology." Pet. Ex. 67 at 3.

Dr. Hawse concluded that the specific molecular mimics and theories provided by Dr. Akbari were "unlikely" to cause Petitioner's disease. Resp. Ex. F at 12. Dr. Hawse opined that Dr. Akbari failed to identify a component of the vaccine that could "trigger an autoimmune response or break immune tolerance to cause injury to [P]etitioner." Id.

In his second report, Dr. Hawse addressed a Dr. Akbari's theory that a "second or possibly third signal is needed to induce autoimmunity" via molecular mimicry. Resp. Ex. F at 12 (quoting Pet. Ex. 110 at 22). Dr. Hawse characterized Dr. Akbari's Treg depletion theory as the proposed second or third signal. Id. Dr. Hawse agreed that the depletion of Tregs could have

[82] Nathan Karin et al., Reversal of Experimental Autoimmune Encephalomyelitis by a Soluble Variant of a Myelin Basic Protein Epitope: T Cell Receptor Antagonism and Reduction of Interferon γ and Tumor Necrosis Factor α Production, 180 J. Experimental Med. 2227 (1994).

adverse effects. Id. at 10. However, Dr. Hawse disagreed that reduced Treg or reduced Treg functionally caused Petitioner's injury. Id. at 12. Further, Dr. Hawse opined it was "improbable" that the flu vaccine reduced Tregs in Petitioner causing her injury. Id. at 8. Additionally, Dr. Hawse opined that there is no evidence that Petitioner had a genetic mutation or any other alternation that would lead to impaired Treg function or generation. Id. at 10. Petitioner did not receive therapy that would deplete Tregs nor was there evidence that Petitioner was "in a state where she severely lacked Tregs." Id. Accordingly, Dr. Hawse found comparisons to studies in which the animal models had depleted Tregs and "an extremely altered immune state" to be "illogical." Id.

Next, Dr. Hawse opined on various inflammasome and cytokine theories put forward by Dr. Akbari. See Resp. Ex. F at 2-7 (discussing and criticizing Dr. Akbari's inflammasome and cytokine theories based on his cited medical literature and criticizing Dr. Akbari's characterization of the medical literature). Dr. Hawse rejected the idea that "high levels of proinflammatory cytokines are generated in response to flu vaccination." Id. at 12. Dr. Hawse opined that it was "unlikely" that the flu vaccine via hemagglutinin and Il-23 cytokine caused a strong Th17 cytokine response resulting in Petitioner's injury. Resp. Ex. C at 8. While Dr. Hawse acknowledged that Il-23 causes Th17 development, he explained that the flu vaccine received by Petitioner does not contain Il-23. Id.

Dr. Hawse argued that case reports do not provide "scientific or epidemiological evidence" of a link between vaccination and TM and opined that no epidemiological studies have established a link between vaccination and TM. Resp. Ex. C at 8. In support, he cited a single study by West et al.,[83] who asserted that "case reports cannot establish a cause-effect relationship" and concluded "the proposed association between vaccination and myelitis is most likely coincidental." Resp. Ex. C-10 at 12-13.

Finally, addressing Dr. Akbari's concerns regarding Baxter et al., Dr. Hawse asserted that the Baxter et al. study primarily received funding from the Center for Disease Control and concluded that journal editors and the peer review have processes to review perceived conflicts of interest when authors receive unrelated grants from vaccine manufacturers. Resp. Ex. C at 8-9; Resp. Ex. F at 1. Further, he noted that at least one article relied on by Dr. Akbari was also sponsored by a vaccine manufacturer. Resp. Ex. F at 11 (citing Pet. Ex. 137).

iii. Althen Prongs Two and Three

Dr. Hawse did not provide opinions on Althen prongs two or three. He limited his opinions to responding to "the immunological merits of Dr. Akbari's theory of causation." Resp. Ex. C at 2.

[83] Timothy W. West et al., Acute Transverse Myelitis: Demyelinating, Inflammatory, and Infectious Myelopathies, 32 Seminars Neurology 97 (2012).

IV. DISCUSSION

A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, a petitioner must prove that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

"Regardless of whether the burden ever shifts to the [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 (2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [P]etitioner's evidence on a requisite element of the [P]etitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

B. Factual Issues

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. § 11(c)(2). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as "the results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." § 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (noting it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such a determination is evidenced by a rational determination).

Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013).

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. Lowrie v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. Cucuras, 993 F.2d at 1528; see also Murphy, 23 Cl. Ct. at 733 ("It has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight." (citing United States v. U.S. Gypsum Co., 333 U.S. 364, 396 (1947))), aff'd, 968 F.2d 1226 (Fed. Cir. 1992).

However, there are situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell v. Sec'y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); Lowrie, 2005 WL 6117475, at *19 ("Written records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting Murphy, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley, 991 F.2d at 1575.

C. Causation

To receive compensation through the Program, Petitioner must prove either (1) that she suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that she received, or (2) that she suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because Petitioner does not allege she suffered a Table Injury, she must prove a vaccine she received actually caused her injury. To do so, Petitioner must establish, by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. Petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on her assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether Petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The special master must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in her favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

V. DIAGNOSIS ANALYSIS

As Federal Circuit precedent establishes, in certain cases it is appropriate to determine the nature of an injury before engaging in the Althen analysis. Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1346 (Fed. Cir. 2010). Since "each prong of the Althen test is decided relative to the injury," determining facts relating to the claimed injury can be significant. Id. Here, the factual issue of diagnosis is in dispute.

The undersigned finds Petitioner provided preponderant evidence to support a diagnosis of TM for the reasons discussed below.

To recap the parties' positions as to diagnosis, Petitioner argued that she suffered from a CNS demyelinating illness, specifically TM and optic neuritis. Pet. Suppl. Br. at 5. Respondent disagreed and asserted Petitioner did not establish by preponderant evidence that she suffered from TM or optic neuritis, and further asserted that Petitioner failed to show "that she was diagnosed with a compensable injury." Resp. Suppl. Br. at 4.

The records establish that Petitioner had a very complex clinical course. She saw many specialists and was given a number of different diagnoses. During Petitioner's initial hospitalization in December 2018, neurologist Dr. Sedarous opined that her symptom of saddle paresthesias was not supportive of GBS. Pet. Ex. 10 at 117-18. During her hospitalization at Jersey Shore Medical Center in December 2018, neurologist Dr. Deutsch noted Petitioner's normal EMG, normal CSF, and normal MRI studies and opined there was no evidence of TM, MS, GBS, or peripheral neuropathy on objective testing. Pet. Ex. 9 at 86-87. But on December 13, 2018, neurologist Dr. Corti expressed concern about GBS "due to active EBV" and ordered IVIG. Pet. Ex. 3 at 31. And, in January 2019, after Petitioner had relapsing paresthesias, neuroimmunologist Dr. Dalakas diagnosed Petitioner with CIDP. Pet. Ex. 11 at 9-14.

A turning point away from a peripheral neuropathy diagnosis (GBS, CIDP) toward a CNS diagnosis occurred mid-January 2019, when Petitioner's MRI showed a thoracic spinal cord lesion at T7/T8, although enhancement was "questionable" due to movement artifact. Pet. Ex. 8 at 209. The radiologist's diagnostic considerations included the question of a demyelinating plaque as seen in focal myelitis or MS. Id. With this study suggestive of a thoracic lesion, Dr. Corti diagnosed Petitioner with TM, attributable to EBV. Pet. Ex. 3 at 15-16, 21.

In April 2019, Petitioner saw neurologist Dr. Pardo-Villamizar at the TM Center at Johns Hopkins, and he diagnosed "thoracic myelopathy." Pet. Ex. 6 at 7. The diagnosis was not "thoracic myelitis," and other etiologies were questioned, including sarcoidosis, lymphoma, or granulomatous myelopathy. Id. While the records during this time are difficult to follow, it appears that these possible causes of Petitioner's condition were worked up and ruled out.

Petitioner saw an MS specialist, Dr. David Duncan, in September 2019, for evaluation and second opinion. Dr. Duncan diagnosed resolved GBS and an "incidental finding of possible demyelinating lesion" on her thoracic MRI, but he did not diagnose TM. Pet. Ex. 15 at 21. He

also later referenced a diagnosis of "underlying [CIDP]." Id. at 15. Dr. Duncan continued IVIG treatment for management of Petitioner's symptoms.

Moving forward to 2020, Petitioner saw neuro-ophthalmologist Dr. Tamhankar for evaluation of optic neuritis. Dr. Tamhankar opined Petitioner had normal OCT findings, no brainstem lesions on MRI, and normal examination. Pet. Ex. 25 at 3,6, 10-11. MRI of the eye orbits done October 2020 showed no optic neuritis. Pet. Ex. 28 at 8-9. In October 2020, Dr. Duncan opined that Petitioner's optical symptoms were of "unclear etiology." Pet. Ex. 29 at 84, 90. Thus, after a workup by a specialist, there does not appear to be treating physician support for the diagnosis of optic neuritis.

The next turning point occurred in December 2020, when Petitioner's treating physicians began to opine that her diagnosis was not clear. When Petitioner saw Dr. Gilson in December 2020, he opined Petitioner "presents a diagnostic dilemma as far as the definitive diagnosis." Pet. Ex. 27 at 10. In March 2021, Dr. Duncan stated Petitioner "has no clear diagnosis at this time." Pet. Ex. 29 at 73.

This lack of clear diagnosis continued in 2022. In Dr. Gilson's records from February 22, 2022, he documents that Dr. Duncan was questioning whether Petitioner's diagnosis was "actually MS." Pet. Ex. 42 at 2. However, Dr. Duncan's records dated March 22, 2022 do not reflect a diagnosis of MS. Instead, Dr. Duncan opined that Petitioner had recurrent EBV infections and he wondered whether the small focal cord abnormalities at T7/T8 and those more recently seen at T10 could represent areas of demyelination as compared to July 9, 2020. Pet. Ex. 42 at 45-46. Thus, Dr. Duncan questioned whether these findings represented thoracic demyelinating lesions. In October 2022, Dr. Duncan wrote, there "is no evidence to make the diagnosis of [MS] at this time" and noted "at present time no clear diagnosis." Id. at 8. This opinion does not appear to be a rejection of his earlier statement that the small focal abnormalities in the thoracic cord could represent demyelination.

In summary, Petitioner's earlier primary treating neurologist Dr. Corti made a diagnosis of TM, but her later neurologist, Dr. Duncan, consistently referenced two peripheral demyelinating conditions, GBS and CIDP, and questioned the diagnosis of TM. Then in 2020, Dr. Duncan, questioned TM, but opined that Petitioner had no clear diagnosis. In light of the evolving clinical course, Dr. Duncan's statement that Petitioner did not have a clear diagnosis appears to be made in the context of monitoring for MS. But the fact that Petitioner did not meet the diagnostic criteria for MS does not negate her earlier diagnosis of TM by Dr. Corti, or Dr. Duncan's concern about the thoracic lesions representing demyelinating plaques.

Here, the undersigned gives more weight to the statements of Petitioner's treating physicians as they are "in the best position" to determine Petitioner's injury. See Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326; Cucuras, 993 F.2d at 1528 (noting contemporaneous medical records, "in general, warrant consideration as trustworthy evidence").

Dr. Ghacibeh opined that Petitioner had NMO based on her MRI findings at T7 and her reversible episode of optic neuritis. Dr. Mackay was persuasive in establishing that Petitioner

did not meet the criteria for a diagnosis of optic neuritis and thus did not meet the diagnostic criteria for NMO. Therefore, the undersigned disagrees that Petitioner had NMO.

The undersigned finds that the weight of the evidence shows that Petitioner was diagnosed and treated for TM, a CNS demyelinating disease. Subsequently, there was a question of whether Petitioner's condition had evolved in the context of monitoring her for MS, and she was not given a diagnosis. The undersigned further finds that the lack of a clear diagnosis in 2022 does not negate Petitioner's prior diagnoses of TM.

Although the undersigned finds that Petitioner carried a diagnosis of TM, this Decision does not turn on a specific diagnosis but instead is based on causation. For example, even if Petitioner's more appropriate diagnosis was a peripheral demyelinating condition like CIDP, or the CNS demyelinating condition NMO, the undersigned would find that Petitioner had not met her burden by preponderant evidence. This is due to the fact that the medical record evidence shows Petitioner had EBV infections and that her treating physicians attributed the cause of her illness to her recurrent EBV infections.

VI. CAUSATION ANALYSIS

A. Althen Prong One

Under Althen prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 257 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If Petitioner relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen, 618 F.3d at 1347 ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

Here, several of Petitioner's treating physicians opined that the cause of her illness was EBV, she tested positive for EBV, and she was noted to have recurrent EBV infections. Further, there is medical literature support for EBV and its causal role in demyelinating illnesses. Due to these facts, the undersigned's determination as to causation turns on an analysis of Althen prong two. Even assuming Petitioner provided evidence of a sound and reliable causal mechanism under Althen prong one, the undersigned finds Petitioner has not provided preponderant evidence of a logical sequence of cause and effect because the evidence establishes she had an EBV infection prior to the onset of her illness. Thus, the undersigned turns her focus to Althen prong two. See Vaughan ex rel. A.H. v. Sec'y of Health & Hum. Servs., 107 Fed. Cl. 212, 221-22 (2012) (finding the special master's failure to rule on Althen prong one not fatal to his

decision because Althen prong two was fatal to the petitioner's case); Hibbard v. Sec'y of Health & Hum. Servs., 698 F.3d 1355, 1364 (Fed. Cir. 2012) ("discern[ing] no error in the manner in which the special master chose to address the Althen [prongs]" when he focused on Althen prong two after "assuming the medical viability of [the] theory of causation").

B. Althen Prong Two

Under Althen prong two, Petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

A petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In determining whether Petitioner has established a prima facia case, the undersigned finds it relevant to consider "evidence of other possible sources of injury" in determining "whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question." Stone, 676 F.3d at 1379. "In asserting an off-Table injury, [Petitioner] need[s] to show, by preponderant evidence, that his vaccination was a substantial factor" in causing her alleged vaccine injury. Winkler v. Sec'y of Health & Hum. Servs., 88 F.4th 958, 962 (Fed. Cir. 2023). Petitioner "[does] not need to show that [she] did not suffer from an infection, or that said infection did not contribute to [her alleged injury]. Nor [does] [she] have to show that the vaccination was the only cause" of her vaccine-related illness. Id.; see also Walther v. Sec'y of Health & Hum. Servs., 485 F.3d 1146, 1149-52 (Fed. Cir. 2007) (finding a petitioner does not bear the burden of eliminating alternative independent potential causes). Thus, the undersigned considers evidence relating to whether Petitioner suffered from an EBV infection, as well as the likelihood that said infection triggered Petitioner's alleged vaccine-related illness, as "[s]uch contemplation of a potential causative agent when evaluating whether or not a petitioner has established a prima facie case is in accordance with the law." Winkler, 88 F.4th at 963; see also Flores, 115 Fed. Cl. at 162-63 ("[T]he special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case.").

Moreover, in evaluating whether Althen prong two is satisfied, the opinions and views of Petitioner's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" (quoting Althen, 418 F.3d at 1280)).

There are three reasons why the undersigned contemplates EBV as a potential cause of Petitioner's demyelinating condition: (1) evidence of positive laboratory evidence of an EBV infection, (2) the opinions of Petitioner's treating physicians, and (3) references in the medical literature.

During Petitioner's clinical course, diagnostic testing showed elevated EBV antibody titers indicating past infection. On December 12, 2018, Petitioner had elevated EBV IgG antibodies at > 750 (normal range 0.0 to 21.9), and elevated EBV nuclear antigens elevated at 34.2 (normal range 0.0 to 21.9). Pet. Ex. 10 at 175-77. The following year, September 14, 2019, testing against showed elevated EBV IgG antibodies at > 750.00 and EBV nuclear antigens at > 600, suggesting past infection. Pet. Ex. 15 at 25-26.

In addition to diagnostic evidence showing that Petitioner had been infected with EBV, her treating physicians documented opinions associating her EBV infection with her demyelinating condition. On December 13, 2018, Dr. Corti opined that Petitioner's presentation was "concerning for [GBS] due to active EBV." Pet. Ex. 3 at 31. On January 15, 2019, Dr. Corti diagnosed Petitioner with TM "with whole body rash, most likely from EBV." Id. at 21-22. The following month, on February 28, 2019, he documented acute TM, "possibly from EBV." Id. at 15-16. Approximately eight months later, Dr. Corti noted that Petitioner had a "whole body rash, possibly from EBV," and that her diagnosis was acute TM and infectious mononucleosis. Id. at 3-4. On February 22, 2022, Dr. Gilson opined Petitioner had "probable MS, EBV [positive] titers." Pet. Ex. 42 at 2-4. In February, March, and October 2022, Dr. Duncan noted Petitioner had "[r]ecurrent EBV infections" in his records. Pet. Ex. 41 at 42-46.

Medical literature filed by the parties establishes that EBV is associated with CNS demyelinating conditions, including TM. Karussis and Petrou state that "the most common type of myelitis worldwide is infectious myelitis." Pet. Ex. 31 at 6. And infectious myelitis is caused by viruses or bacteria, including EBV. Id. Erdem et al. reported that TM "usually occurs as a post-infectious complication and appears to result from an autoimmune process." Pet. Ex. 39 at 1-2. Langer Gould et al. reported that a history of infectious illnesses was present in 90 cases (11.5%) of the of newly diagnosed patients with MS, clinical isolated syndrome, or ADEM. Pet. Ex. 35 at 3, 4 tbl.1. Huynh et al. reported that EBV was associated with ADEM. Pet. Ex. 33 at 5.

Petitioner filed three recent studies identifying an association between EBV and MS. See Pet. Ex. 67 at 1 (findings "suggest EBV as the leading cause of MS"); Pet. Ex. 68 at 1 ("[R]esults provide a mechanistic link for the association between MS and EBV . . . ."); Pet. Ex. 69 at 3 ("[T]he evidence that EBV has an early causal role in the development of MS is most compelling.").

The experts' opinions lined up with their respective parties' positions. Dr. Ghacibeh disagreed that Petitioner's EBV was relevant, arguing that most people have antibodies against the virus. Dr. Akbari acknowledged that EBV is "the leading cause" of demyelinating disease relevant to the mechanism of molecular mimicry, although he also used this example of molecular mimicry to support his opinion that the Petitioner's flu vaccination caused her TM. Pet. Ex. 45 at 13-15.

Respondent's expert, Dr. Bromberg opined that EBV was a "possible cause" of Petitioner's symptoms, noting that she had antibodies to EBV and several episodes of EBV infections. Resp. Ex. A at 12. He also cited the opinions of the treating physicians relative the EBV. Dr. Hawse also acknowledged the molecular mechanism link between EBV and MS. He criticized Dr. Akbari's attempt to use this example of molecular mimicry to extrapolate to the context of the flu vaccine and demyelinating disease, especially MS. The undersigned finds Dr. Hawse's opinions regarding this point more coherent and persuasive.

In summary, the undersigned finds preponderant evidence that Petitioner had an EBV infection, that her treating physicians documented opinions noting a causal association between her EBV infection and demyelinating illness (whether diagnosed as GBS, CIDP, or TM), and that this causal association is supported by medical literature. The undersigned acknowledges that Petitioner is not required to eliminate other potential causes in order to be entitled to compensation. See Walther, 485 F.3d at 1149-52 (finding petitioner does not bear the burden of eliminating alternative independent potential causes). However, she finds it reasonable to consider "evidence of other possible sources of injury"—here, Petitioner's EBV infection—in determining "whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question." Stone, 676 F.3d at 1379.

In Winkler, the Federal Circuit recently affirmed the undersigned's reliance on "evidence of other possible sources of injury"—in Winkler, a GI illness—in determining "whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question." 88 F.4th at 962-63 (quoting Stone, 676 F.3d at 1379). Like Petitioner here, the petitioner in Winkler had an infectious illness (diarrhea) prior to developing symptoms of a demyelinating illness. Id. at 961. Here, like in Winkler, all experts acknowledge a "potential causative agent," which here, is that the Petitioner had EBV and that her EBV was a possible source of TM (or by some treating physician accounts a likely source of Petitioner's illness). Id. at 963. The undersigned dismissed the petition in Winkler because she found petitioner did not provide preponderant evidence that the vaccine was a substantial factor in causing his GBS. Id. at 961. The Federal Circuit affirmed, noting "contemplation of a potential causative agent when evaluating whether or not a petitioner has established a prima facie case is in accordance with the law." Id. at 963.

Therefore, the undersigned finds Petitioner has not proven by preponderant evidence a logical sequence of cause and effect establishing that the flu vaccination caused Petitioner's demyelinating illness. Thus, Petitioner has not satisfied the second Althen prong.

C. Althen Prong Three

Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That phrase has been defined as a "medically acceptable temporal relationship." Id. A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically

acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen prong one). Id.; see also Koehn v. Sec'y of Health & Hum. Servs., 773 F.3d 1239, 1243-44 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. 532,542 (2011). Thus, prong three contains two parts. First, Petitioner must establish the "timeframe for which it is medically acceptable to infer causation" and second, they must demonstrate that the onset of the disease occurred in this period. Shapiro, 101 Fed. Cl. at 542-43.

Petitioner received the flu vaccination on November 8, 2018. The onset of her ascending paresthesias began on December 3, 2018. Respondent agrees this time frame for onset is medical acceptable, stating that "Petitioner's neurologic symptoms began twenty-five days post-vaccination and [] a medically acceptable timeframe following the flu vaccination." Resp. Prehrg. Br. at 26 (citing Pet. Ex. 10 at 117).

Dr. Ghacibeh opined that this 25 day onset is consistent with reports stating that acute central demyelination usually occurs within 30 days of vaccination. Dr. Akbari agrees. Respondent's experts do not take issue with the onset time frame, although they dispute that the Petitioner's sensory symptoms were associated with TM or caused by vaccination.

The time frame of 25 days is an appropriate onset acknowledged in other Vaccine Program cases in which molecular mimicry has been proffered as the causal mechanism. See, e.g., Simeneta v Sec'y of Health & Hum. Servs., No. 18-859V, 2024 WL 4881411, at *34-35 (Fed. Cl. Spec. Mstr. Oct. 31, 2024) (finding a GBS onset of 18 days after Prevnar 13 vaccination to be appropriate); Bartoszek v Sec'y of Health & Hum. Servs., No. 17-1254V, 2024 WL 4263604, at *24-25 (Fed. Cl. Spec. Mstr. Aug. 27, 2024) (finding a GBS onset of 22 days, or approximately three weeks, post-Prevnar 13 vaccination to be medically acceptable); Kelley v. Sec'y of Health & Hum. Servs., 68 Fed. Cl. 84, 102 (2005) (finding CIDP onset approximately two weeks after flu vaccination acceptable); Daily v Sec'y of Health & Hum. Servs., No. 07-173V, 2011 WL 2174535, at *9 (Fed. Cl. Spec. Mstr. May 11, 2011) (finding that onset of CIDP within a few weeks of flu vaccination was a medically acceptable timeframe); Introini v. Sec'y of Health & Hum. Servs., No. 20-176V, 2022 WL 16915818, at *27-28 (Fed. Cl. Spec. Mstr. Oct. 19, 2022) (finding TM onset approximately five weeks after vaccination medically acceptable); see also Barone v. Sec'y of Health & Hum. Servs., No. 11-707V, 2014 WL 6834557, at *13 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) ("[S]pecial masters have never gone beyond a two-month (meaning eight week) interval in holding that a vaccination caused a demyelinating illness.").

However, a temporal association, without more, is insufficient. Moberly, 592 F.3d at 1323; Grant v. Sec'y of Health & Hum. Servs., 956 F.2d 1144, 1148 (Fed. Cir. 1992) ("[A] proximate temporal association alone does not suffice to show a causal link between the vaccination and the injury."). Thus, even though Petitioner has provided preponderant evidence satisfying Althen prong three, Petitioner is not entitled to compensation.

VII. CONCLUSION

The undersigned extends her sympathy to Petitioner for all that she has suffered. The undersigned's Decision, however, cannot be decided based upon sympathy, but rather on the evidence and law.

For the reasons discussed above, the undersigned finds that Petitioner has failed to establish by preponderant evidence that the flu vaccination she received caused her to develop TM, optic neuritis, or any other alleged conditions. Therefore, Petitioner is not entitled to compensation and the petition must be dismissed.

In the absence of a timely filed motion for review pursuant to Vaccine Rule 23, the Clerk of Court **SHALL ENTER JUDGMENT** in accordance with this Decision.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master